CONTRACEPTIVE USE AND CONTROLLED FERTILITY

Health Issues for Women and Children

Background Papers

Allan M. Parnell, editor

Working Group on the Health Consequences
of Contraceptive Use and Controlled Fertility

Committee on Population
Commission on Behavioral and Social Sciences and Education
National Research Council

NATIONAL ACADEMY PRESS
Washington, D.C. 1989

NATIONAL ACADEMY PRESS • 2101 Constitution Avenue, NW • Washington, DC 20418

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Library of Congress Catalog Card No. 89-63018
International Standard Book Number 0-309-04096-5

Additional copies of this report are available from:

National Academy Press
2101 Constitution Avenue, N.W.
Washington, D.C. 20418

S030

Printed in the United States of America

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Preface

The four papers in this volume are the result of work carried out by a Committee on Population–sponsored Working Group on the Health Consequences of Contraceptive Use and Controlled Fertility. The Committee on Population was asked to undertake an assessment of the likely health consequences to women and their children of changes that were under way or anticipated in many developing countries in the number of children, the timing between them, and the age at which women were giving birth. A working group, chaired by William Foege and Julie DaVanzo, investigated this topic. The result of their assessment, *Contraception and Reproduction: Health Consequences for Women and Children in the Developing World,* has been published by the National Academy Press in a separate volume.

In the course of completing their assessment, the working group commissioned a series of background papers dealing with different aspects of the relationships among changing reproductive patterns, contraceptive use, and the health of women and children. A complete list of these papers is given in the appendix to this report. In addition, the working group also benefited from a series of special analyses undertaken on its behalf by scholars with a special interest in the topic of fertility and health.

Unfortunately, it was not possible to publish all of the papers and analyses commissioned by the working group. Some of the work resulted in only a set of tabulations that members of the working group themselves incorporated into the report and that would not be appropriate for separate publication. Other papers were highly specialized and judged to be more appropriate for publication in scientific journals. There were, however, a series of papers that provided important background information for the report and that brought together data and analyses that are otherwise difficult to obtain in a single source. Two of the selected papers combined biomedical and demographic perspectives in a particularly useful way, while a third provides an unusually comprehensive overview of technical issues of great concern to those interested in fertility and health. The fourth suggests a framework for thinking about the psychosocial consequences to women of contraceptive use and controlled fertility. The Committee on Population decided to publish these four papers.

The first paper, by Susan Zimicki of the University of Pennsylvania, deals with the relationship between changing reproductive patterns and women's health. The second paper, by Nancy Lee, Herbert B. Peterson, and Susan Y. Chu of the Centers for Disease Control, provides an overview of the risks and benefits of different modern contraceptives. The third paper, by John G. Haaga of the Rand Corporation, reviews the evidence on the relationship between changing reproductive patterns and child health. In addition, a more specialized paper is included, which in the judgment of the working group, brings together reports of particularly important aspects of the relationship between health and fertility and merits wider consideration than it might otherwise receive. This paper, by Ruth Dixon-Mueller of the University of California, Berkeley, deals with psychological aspects of changing reproductive patterns. The working group's report, like almost all the other literature in this field, deals almost entirely with physical health; nevertheless, the working group recognizes that there are important psychological and mental health aspects to human reproduction that to date have not been carefully evaluated. It is our hope that this paper will encourage more careful thinking about this topic.

The Committee on Population and the Working Group on the Health Consequences of Contraceptive Use and Controlled Fertility are very grateful for the work completed on its behalf by the authors of these papers as well as by those who prepared the other papers and analyses listed in the appendix. In addition, the committee and the working group wish to thank the Agency for International Development and the Rockefeller Foundation for providing financial support for the completion and publication of its report and these papers.

The Committee on Population and the working group are also grateful to the staff members who worked on this project, including Peter J. Donaldson, study director; Susan Rogers, research associate; Diane Goldman, administrative secretary; and Eugenia Grohman, associate director for reports of the Commission on Behavioral and Social Sciences and Education. Special thanks are due Allan Parnell, who served as the principal staff officer for the Working Group on the Health Consequences of Contraceptive Use and Controlled Fertility and as editor of this volume of papers.

ALBERT I. HERMALIN
Chair, Committee on Population

The Relationship Between Fertility and Maternal Mortality

Susan Zimicki

Maternal mortality is much higher in developing than in developed countries (Mahler, 1987). This is clearly a function of a number of factors, including the greater risk inherent in pregnancy and delivery owing to lack of adequate medical care; the greater prevalence of infectious diseases, which are cofactors in some deaths; and the higher incidence of pregnancy. Because high-mortality countries are those with the least reliable vital statistics, little information is available about levels and risk factors.

Provision of family planning services has been proposed as one way to reduce maternal mortality (Rosenfield and Maine, 1985). The argument is that use of family planning services will reduce the absolute number of pregnancies and will allow shifts in the timing of pregnancy from high-risk to lower-risk ages and from shorter to longer interbirth intervals.

This paper will review the information available about the effects of parity, age, and birth intervals on maternal mortality and morbidity, with particular attention to some of the common complications of pregnancy and the major causes of death. Winikoff and Sullivan (1987) have examined limits of the possible effect of family planning programs in reducing maternal mortality, and Trussell and Pebley (1984) have quantified the possible impact of changing age and parity distributions on fertility. In addition to including more recently available population-based information, this paper considers in detail some of the

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major mechanisms through which maternal mortality occurs and how they are related to fertility.

DEFINITIONS AND MEASUREMENT ISSUES

Measures

A number of different measures of maternal mortality are commonly used, the most important being the maternal mortality ratio and the maternal mortality rate. The numerator for both is the same: the number of maternal deaths occurring in a given period. Most studies adhere to the International Classification of Disease, ninth revision (ICD-9), definition of maternal death in including the deaths of women within 42 days of termination of pregnancy. Some older studies may include deaths that occurred up to 90 days after the termination of pregnancy, in accordance with the Guide of Maternal Deaths Studies published by the American Medical Association (1964).

The maternal mortality ratio is the ratio of maternal deaths per *live births* (variously 1,000, 10,000, or 100,000; unless noted otherwise a metric of 1,000 is used for ratios and rates in this paper). Although the denominator should ideally be pregnancies, the impossibility of obtaining accurate counts of fetal losses and stillbirths has necessitated using live births. According to the World Health Organization (WHO) (1985), in countries with low induced-abortion rates, the number of live births is within 10 percent of the number of pregnancies. This ratio measures the probability of maternal death, the *obstetric risk*.

The maternal mortality rate is simply a cause-specific death rate: maternal deaths/women of reproductive age (variously 10 to 49, 15 to 44, 15 to 49 years old). This rate is a function of the incidence of pregnancy as well as the risk inherent in pregnancy. Thus, the maternal mortality rate is linked to the maternal mortality ratio through the general fertility rate (births/1,000 women of reproductive age).

Two extensions of the maternal mortality rate have been used as measures of maternal mortality: (1) the proportion of all-cause mortality for women of reproductive age that is attributable to maternal mortality and (2) the lifetime risk of maternal mortality, calculated as reproductive-span maternal mortality rate (Measham and Herz, cited in Fortney, 1987).

It is important to consider the effects of age and fertility structure differences on these measures. Obstetric risk varies greatly with age, as does the incidence of pregnancy. Thus, populations that have different age structures and/or different age-specific fertility rates may have different crude maternal mortality ratios even when they have similar age-specific ratios (see Fortney, 1987, and Graham and Airey, 1988, for examples). Whether or not the crude ratios are different depends on whether the ages of highest obstetric risk are those in which the population or fertility differences reside. The crude maternal mortality rate and the measures based on it are even more sensitive to these structural differences.

Unfortunately, nearly all studies report only crude maternal mortality ratios and rates, making comparisons across populations or even of the same population at successive times questionable.

Sources of Data

Maternal mortality is underestimated even in countries with excellent vital registration systems; official statistics from countries where maternal mortality is high seriously underestimate the true level (WHO, 1987). Better data are available from three sources: population-based studies, hospital population studies, and case series. Population-based studies provide the least biased estimates of maternal mortality. They allow nearly complete counts of live births and maternal deaths. Because of their prospective nature they include deaths that occur more than a week after delivery and probably include most of the deaths due to induced abortion. There are very few population-based studies concerning maternal mortality: six from Bangladesh (Chen et al., 1975; Lindpaintner et al.,1982; Khan et al., 1986a; Alauddin, 1987; Koenig et al., 1988; Faveau et al., 1988); one from Ethiopia (Kwast et al., 1986); one from Egypt (Fortney et al., 1985);[1] one from the Gambia (Greenwood et al., 1987); and one from Jamaica (Walker et al., 1985). Preliminary results from a population-based study in India are available (Bhatia, 1985). Although the Medical Research Council (MRC) project in the Gambia and the Machakos project are population laboratories that have yielded excellent information on a number of topics, maternal mortality in both areas has been almost completely eliminated because of care provided by the project (Lamb et al., 1984; Voorhoeve et al., 1979), and so these studies are not included. In addition, one hospital-based study from Lusaka, Zambia (Mhango et al., 1986), arguably covers a sufficient portion of the population (85 percent of births, 90 percent of deaths) to be considered a population-based study. Unfortunately, the number of deaths in each study is generally very small, so estimates by specific cofactors are unstable.

With regard to morbidity the WHO collaborative studies on family formation patterns and health provide population-based information about anemia and uterovaginal prolapse in nine countries (Omran et al., 1981a, 1981b). In addition, one study from Jerusalem (Harlap et al., 1971) concerns obstetric interventions, and one from the Philippines (Raymundo, 1987) concerns morbidity during the last pregnancy.

Hospital population studies, particularly those from referral hospitals, provide less accurate estimates of the incidence of maternal mortality or morbidity than do population-based studies. They reflect the experience of only a proportion of the population during only part of the risk period at the time of and immediately after pregnancy outcome. Many of the hospital-based studies report only the crude maternal mortality ratio and limit further analyses to the series of deaths. A few report case-fatality rates for various complications. If the assumption can be made that the deaths in hospitals are representative of all maternal deaths or the way in which they are unrepresentative can be identified, then some examination of the proportional contribution of various causes becomes worthwhile.

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[1] The companion study to the one carried out in Egypt was carried out in Bali. It is not included as a population-based study because the ascertainment rate of deaths was estimated as 50 percent at most. With that level of underestimation, it seems unlikely that the information is representative.

Case series—reports about a series of deaths, either due to any maternal cause or to a specific cause, such as ruptured uterus, eclampsia, or hepatitis—can provide valuable information about incidence and case-fatality rates. Unfortunately, most contain no description of the population from which the cases are drawn. However, series selected because of the presence of a risk factor (e.g., the Arkutu, 1978, series of primigravidae) rather than an outcome and those in which the maternal deaths are compared to a subsample of the hospitalized population represent one of the most efficient ways of obtaining information about maternal mortality.

Problems of Measurement

As noted above, the maternal mortality ratio as defined by the WHO is already an approximation of the measure of interest. However, the more serious problems in measurement arise from incomplete ascertainment of either the number of deaths or the number of related live births. Deaths that occur before delivery, such as those due to ectopic pregnancy, may not be recognized as maternal deaths. Additionally, those due to induced abortion may be misrepresented because of shame or fear of prosecution: in one series of cases of women who came to a hospital with tetanus "all [22] postabortal cases followed criminal abortion, although in only 10 cases was the interference admitted by patients or their relations" (Adadevoh and Akinla, 1970). Another group of deaths that is easily missed, especially in hospital-based studies, are those that occur some time after delivery. The population-based study in Tangail (Alauddin, 1987) shows 15 percent and that in Jamalpur (Khan et al., 1986a) shows 27 percent of deaths occurring more than a week after delivery.

Depending on the time period of interest, retrospective population-based studies may underestimate both maternal deaths and live births. A valid count of live births is generally less of a problem for prospective population-based studies but exists nevertheless. Comparison of the crude birth rate for the Jamalpur prospective study with the national rate suggests an underreporting of up to 10 percent of the births (Khan et al., 1986a); the same comparison for the Tangail prospective study suggests underreporting of 27 percent (Alauddin, 1987).

The problem of estimation of the denominator is most crucial, however, with regard to hospital-based studies. In developing countries many women do not usually deliver in hospitals, although they may be brought there if the delivery is complicated. Hospital-based maternal mortality ratios in urban areas with easy access are probably overestimates because a larger proportion of more difficult than normal deliveries is represented among deliveries. This tendency for hospital populations to overrepresent abnormal or complicated cases is exacerbated in the case of referral or teaching hospitals in large cities, which receive not only all the self-referred emergency deliveries but also patients referred by other health facilities. As most hospital reports are generated by teaching or referral hospitals, the reported maternal mortality ratios are likely to be overestimates. One study reported two ratios, the first 16.7, calculated on the basis of hospital births, the second, 3.9, on the basis of births in the city (Rao, 1975).[2] Unfortunately, the number of births in the hospital catchment population often cannot be ascertained or estimated; this might happen, for example, if there are several hospitals in a city. The representativeness of the hospital population can be assessed using two observations that are frequently reported: the proportion of admissions that are emergency or unbooked and the proportion of deaths that occur soon after admission.

In areas where access to a hospital is restricted (because of distance, cost, or social barriers), a greater proportion of women experiencing difficulty during delivery will die at home. Ratios reported by these hospitals may represent underestimates; in addition to the advantage of medical care at the time of delivery, the population delivering in hospitals may have had greater than average exposure to antenatal care and may represent a more socioeconomically advantaged portion of the population. One extreme example is from the Medical Research Council (MRC) study area in the Gambia: the maternal mortality ratio for the period 1951-1975 was reported as about 10 (Billewicz and McGregor, 1981), but during 8 years after the establishment of a continuous medical service there were no maternal deaths, although 16 would be expected if the established ratio had prevailed (Lamb et al., 1984). An additional problem of smaller rural facilities is that caseloads are often insufficient to produce stable estimates of mortality.

While estimates of maternal mortality ratios from hospital-based studies may be biased, they provide a useful supplement to the few estimates obtained from population-based studies. However, because of the very small numbers of deaths identified in population-based studies,[3] using hospital-based studies is crucial for examination of the causal mechanisms involved in high maternal mortality.

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[2] In the light of the probable large effect of selection bias, the problem of maternal mortality ratios calculated using deliveries rather than live births in the denominator is minor, but a number of hospital reports (and one population-based study: Greenwood et al., 1987) use deliveries. While this is more correct epidemiologically, as it is a better estimate of the population at risk, it deviates from the international definition.

[3] The total number of deaths reported in the nine identified population-based studies is 936; 630 of these come from two countries—Egypt and Jamaica—with the lowest and third lowest reported maternal mortality rates in the series.

If all the maternal deaths from an area occur in a hospital, the proportion of mortality associated with each cause of interest can be determined accurately (apart from classification bias), even when the maternal mortality ratio cannot be determined. This situation probably occurs more frequently when a large proportion of the population is already delivering in a hospital—for example, in Lusaka (Mhango et al., 1986). When it does not occur, three types of deaths are most likely to be excluded. First, many early-pregnancy deaths occur at home: in Addis Ababa 6 of 13 abortion-associated deaths occurred at home. Some hospital studies explicitly exclude all early-pregnancy deaths (e.g., Chi et al.,1981; Hartfield, 1980) as well as those occurring after the woman has been discharged from the hospital, while a few simply do not report any (Balachandran cited in Armon, 1977; D'Cruz et al., 1986a).

The second type of death apt to be omitted from hospital statistics comprises those due to indirect causes—not resulting from the complications of pregnancy itself but rather from a condition that is aggravated by pregnancy or one that is completely unrelated, such as a motor vehicle accident. Kwast et al. (1986) report hospital deaths for 67 percent of those dying of obstetric causes but only 38 percent of those dying of indirect causes. Even if they die in hospitals, women who die of indirect causes may, for example, die in the medical ward and never come to the attention of those who report "maternal mortality." Finally, women are more apt to be seen in a hospital if the delivery complication "affords relatives the time to discuss the merits of hospital admission" (Hartfield, 1980) and the hospital is sufficiently close to the woman's home. One way to estimate this effect is to examine the ratio of deaths from obstructed labor and hemorrhage to those from hypertensive disease; this ratio tends to be much higher in hospital-based studies than in population-based ones.

In addition to these types of inclusion bias, hospital series reports are also subject to classification bias. For example, deaths resulting from cephalopelvic disproportion and abnormal presentations leading to prolonged labor and uterine rupture might be classified as due to ruptured uterus if the rupture is identified, as due to hemorrhage if extravaginal bleeding is a prominent feature, or to sepsis if death is delayed some time after the onset of labor. Many maternal deaths are associated with a number of complications; for example, of 219 deaths in Zaria only 86 could be attributed to a single condition, while the rest were associated with two or more (Harrison and Rossiter, 1985).

This then is the situation: maternal mortality is inherently difficult to measure; the summary measures commonly used obscure any differences between populations that might be due to fertility patterns as opposed to other sources of risk; the best (though still flawed) data come from a few extremely small studies in restricted geographic areas; and the largest source of data is subject to selection and classification biases that cannot easily be controlled for because they are unquantifiable except in the very particular circumstances of each study. Even with these drawbacks, fairly clear patterns are apparent both in terms of the geography of level and causal components and with reference to the relation between fertility and maternal mortality.

WORLD PATTERNS

Population and hospital-based studies from countries selected because they had several studies show distinct differences in the levels of maternal mortality in different regions (Table 1). The highest mortality ratios are from the population-based studies in the Gambia, where the overall ratio is probably between 10 and 20 per 1,000 births. Hospital studies from Nigeria and a population-based study from eastern Senegal (Pison, 1989) suggest that the risk in West Africa is generally high, though perhaps closer to 10 than 20. The next highest mortality occurs in South Asia; from the population studies in Bangladesh and the population and hospital studies in India, a maternal mortality ratio of 4 to 8 seems most likely. Indonesia and Ethiopia probably have similar levels. The information from Tanzania suggests that the risk is slightly lower there, probably around 2 to 4, and the studies from South Africa suggest the same level. The three hospital-based studies in Lusaka, Zambia, are very consistent, and maternal mortality—at least in the city, where rates of prenatal care and hospital delivery are very high (Mhango et al., 1986)—is most likely in the range of 1 to 2.

The ratios now seen in Lusaka are beginning to approach those seen in Latin American countries in the early 1960s. More recent studies have shown ratios 10 times lower.

Thus, if developing country regions are ranked by risk of childbearing, West Africa is clearly the area of highest risk, followed by South Asia, northern East Africa, and southern Africa. Latin America is clearly the region of lowest risk.

ASPECTS OF FERTILITY AS RISK FACTORS FOR MATERNAL MORTALITY

Fertility can be described in terms of age of first occurrence, total number of events, and interval between them. All of these factors are susceptible to family planning interventions; contraception can be used to delay the first pregnancy, lengthen the interval between births, and reduce the total number of pregnancies.

Parity/Gravidity

The event that puts a woman at risk of maternal mortality is conception. Strictly, the relationship between fertility and maternal mortality should be described in terms of pregnancies. Some risks (e.g., hemorrhage from ruptured ectopic pregnancy, complications of induced abortion, amplification of the risk of infectious diseases) accrue to pregnancy well before delivery, while others are

TABLE 1 Maternal Mortality Ratio Per 1,000 Births From Selected Countries

Country	City	Population	Hospital	Year	Reference
Gambia	Keneba	10.5		1951–75	Billewicz and McGregor (1981)
	Mandua	9.5		1951–75	Billewicz and McGregor (1981)
	North Bank	22.0		1981–83	Greenwood et al. (1987)
Ethiopia	Addis		7.8	1980	(Frost) cited in Kwast et al. (1986)
	Addis Ababa	4.6		1981–83	Kwast et al. (1986)
	Addis Ababa		6.9	1981–83	Kwast et al. (1986)
Tanzania	Kilimanjaro		3.2	1971–77	Armon (1979)
	Dar es Salaam		2.1	1974–77	Mtimavalye et al. (1980)
	Moshi		2.6	1980–81	Janowitz et al. (1984)
	Mwanza		2.3	1982	Janowitz et al. (1984)
Zambia	Lusaka		1.5	1974	(Hickey) cited in Hartfield (1980)
	Lusaka		1.6	1974–78	(Narone) cited in Boerma (1987)
	Lusaka		1.2	1982–83	Mhango et al. (1986)
South Africa	Pietermaritzburg		1.5	1973–75	Barford and Parkes (1977)
	Durban		2.1	1975–82	Melrose (1984)
Indonesia	12 hospitals		3.9	1977–80	Chi et al. (1981)
	Bali	7.2		1980–82	Fortney et al. (1985)
Bangladesh	Matlab	7.7		1967–68	Chen et al. (1975)
	Matlab	5.7		1968–70	Chen et al. (1975)
	Matlab	5.1		1982	Lindpaintner et al. (1982)
	Tangail	5.7		1982–83	Alauddin (1987)
	Jamalpur	6.2		1982–83	Khan et al. (1986)
India	Calcutta		8.4	1956–58	Dawn et al. (1972)
	Calcutta		5.9	1966–68	Dawn et al. (1972)
	Madurai	3.9	16.5	1960–72	Rao (1975)
	Bombay urban		4.0	1961–69	Shah et al. (1971)
	Bombay rural		2.4	1961–69	Shah et al. (1971)
	Anantapur rural	8.7		1984–85	Bhatia (1985)
	Anantapur urban	5.5		1984–85	Bhatia (1985)
Colombia	Bogota	1.3		1962–64	Puffer and Griffith (1967)
	Cali	2.2		1962–64	Puffer and Griffith (1967)
Venezuela	Caracas	1.0		1962–64	Puffer and Griffith (1967)
	Cumana		0.3	1978–80	Janowitz et al. (1982a)
Brazil	Ribeirao Preto	0.6		1962–64	Puffer and Griffith (1967)
	Sao Paulo	0.9		1962–66	Puffer and Griffith (1967)
	Campinas		0	1977–79	Janowitz et al. (1982b)
Chile	Santiago	3.2		1962–64	Puffer and Griffith (1967)
	Santiago		0.3	1977–78	Janowitz et al. (1982a)

inherent in delivery itself. Most of the studies concerning maternal mortality report parity-specific mortality; a few report rates by gravidity.[4]

All the population-based studies indicate and results from hospital studies generally confirm that the first birth and births of high order are strong risk factors for maternal mortality (Table 2). These studies indicate a J- or U-shaped risk with parity: high during the first pregnancy, lowest during the second or third, and high again by the fifth pregnancy. A similar pattern is found for gravidity, with an even stronger relative risk for first pregnancies relative to later-order pregnancies than is observed for first births.

Corroboration for the higher risk of first deliveries comes from the population-based morbidity study carried out in West Jerusalem, where the obstetric intervention rate was calculated by parity (Harlap et al., 1971). Obstetric interventions included any specialist intervention at any stage of labor, including surgical or medical inductions of labor, forceps or vacuum deliveries, breech deliveries, cesarean sections, and other major third-stage interventions; while probably an overestimate, this measurement does indicate potential mortality in the absence of medical care. Both the parity-specific rates and the ratios (adjusted for age, ethnicity, and hospital) indicate about a 70 percent higher risk for first than for second births (Table 3).

Except for very high parity births (10+), this study does not confirm the excess risk of multiparity. This is surprising in light of the very clear enhanced risk of malpresentation for multiparous women noted by, for example, Faundes et al. (1974): 3.4 breech presentations per 1,000 women of parity 0, 10.1 for parities 1 and 2, 14.4 for parities 3 and 4, 17.5 for parities 5 and 6, and 19.0 for parity 7+. A review of a series of 50,057 deliveries, including 5,785 to grand multiparous (parity 7+) women in Haifa, Israel (Fuchs et al., 1985), yielded a rate of malpresentation of 11.9 per 1,000 for the grand multiparous women in contrast to a rate of 3.3 per 1,000 for women of lower parity.

A study from the Sudan (Aziz, 1980) comparing 2,049 primiparous, 3,679 multiparous, and 3,130 grand multiparous (5+) women found slightly higher rates of abnormal presentation for grand multiparas (14 percent compared to 9 percent for multiparas and 9.5 percent for primiparas). This same study found more toxemia among primiparas than other women (10.8 percent vs. 7.8 percent for multiparas and 8.5 percent for grand multiparas), more anemia among grand multiparas (7.4 percent) than other women (4.5 percent), and a clear increase in the risk of antepartum hemorrhage with parity (primiparas 1.3 percent, multiparas 1.9 percent, grand multiparas 4.5 percent). Al-Sayegh and Hathout (1974) in Kuwait obtained similar results for antepartum hemorrhage (0.1 percent for women of parities 1 to 5, 1.2 percent for parities 6 and 7, and 4.2 percent for parity 8+), but they observed lower rates of abnormal presentation—only 5.1 percent for

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[4] Parity is the number of previous live births; gravidity is the number of previous pregnancies. Information about parity is probably more accurate than that about gravidity.

TABLE 2 Mortality Per 1,000 Live Births by Parity and Gravidity Prior to Current Pregnancy

Gravidity Parity	Tangail[a] G	Tangail[a] P	Jamalpur[b] P	Matlab[c] G	Matlab[c] P	Matlab[d] G	Matlab[d] P	Addis Ababa[e] P	Jamaica[f] P	Gambia[g] P
0	5.5	3.8	33.8	12.7	9.9	9.7	9.5	13.3	0.8	28.6
1	4.3	3.7	4.2	2.4	2.8	4.1	3.8	8.1	0.6	11.9
2	3.4	4.2		2.6	2.3	3.1	3.1	3.2	0.8	
3	3.8	5.7	7.6	2.4	4.8	3.1	3.4		0.8	
4	7.7	2.3		4.7	4.3	4.9	5.2	5.4	0.9	
5	9.1	9.1	14.2	6.0	5.9	3.4	3.7	3.0	1.3	45.5
6		14.9		6.8	5.2	5.9	4.9			
7	10.8	14.9		7.4	7.2	6.9	6.8			
8				6.5	7.3	8.3	10.4			
9+				6.6	7.8		8.2			
Total	5.7	5.7	6.2	5.7	5.7	5.5	5.5	4.9	1.1	22.3

Note: Close examination of the population-based studies indicates some confusion about the definitions of parity and gravidity. Chen et al. (1975) label groups of women by their status prior to the current pregnancy and report, as might be expected, fewer deaths and live births among women who had never had a pregnancy than among those who had never had a live birth (but who might have had a prior pregnancy). Alauddin (1987) also reports both parity-specific and gravidity-specific ratios, but labels women with no previous live births parity 0 and those with no previous pregnancies gravidity 1. In addition, there seems to be a classification problem, as the reported ratios indicate 10 deaths among women who had no prior pregnancy but only 7 among those with no prior live birth. Walker et al. (1985) have classified women as to parity by considering the number of prior pregnancies lasting at least 28 weeks but not necessarily terminating in a live birth, *including the current pregnancy*. Thus all women who have never had a prior live birth are considered parity 1; in general, even after correction for this difference, the parities of women in the Jamaica study will be slightly inflated relative to reported parities of women in the other studies. This study also includes deaths that occurred up to a year after delivery. In the population-based study from Ethiopia (Kwast et al., 1986), a note to the parity 0 rate indicates that it has been calculated per 1,000 nulliparous women. As the parity 1 through 5+ live births add up to the total number of live births in the study, it seems likely that the parity classification includes the current pregnancy, with deaths to women with no prior live birth being categorized as parity 0 if they did not survive to deliver and parity 1 if they delivered. This rate has been recalculated. Rates reported by Greenwood et al. (1987) are calculated over all pregnancies, as parity-specific numbers of live births are not available.

Sources: [a]Alauddin (1987), [b]Khan et al. (1985), [c]Chen et al. (1985), [d]Koenig et al. (1988), [e]Kwast et al. (1986), [f]Walker et al. (1985), and [g]Greenwood et al. (1987).

women of parity 8 or more. Their case series is, however, quite a bit smaller, including 2,060 women of parities 1 to 5, 494 of parities 6 and 7, and 446 of parity 8+.

In a 1958–1960 multicenter study in the United States, Israel and Blazar (1965) reported a much higher rate of essential hypertension among grand multiparas (7+) (without controlling either for age or race), higher rates of uterine rupture and postpartum hemorrhage, and significantly higher rates of placenta previa and placental abruption, but no difference in maternal mortality, presumably because of adequate hospital care.

Interaction Between Age and Gravidity/Parity

Most information about complication or mortality is couched in terms of either age or parity: women are at greatest risk at young and old ages and at low and high parities. Because age and parity are strongly associated, it is not clear if the age-specific and parity-specific patterns reflect the same basic age-driven risk, if they have independent effects, or if they act in combination. Contraception provides the means of affecting timing of fertility and thus increases the importance of knowing whether there is an interaction between age and parity in their effects on morbidity and mortality associated with fertility.

Four of the population-based studies provide information about maternal mortality by age and parity. The simple J- or U-shaped relationship between parity

TABLE 3 Obstetric Interventions by Parity in Jerusalem

	Intervention	
Parity	Rate/1,000	Ratio
1	244	146
2	138	84
3	145	89
4	116	81
5–6	107	71
7–9	103	73
10+	125	84
All	159	100

Note: Ratio is adjusted for age, ethnic group, and hospital. Overall maternal mortality ratio in this population was 0.03 per 1,000 births.

Source: Harlap et al. (1971).

and maternal mortality is less clear-cut when age is taken into account. Part of the reason for this is the small number of deaths in any of the studies; as a result, the age-parity-specific estimates are fairly unstable, even when some categories are grouped. Two of the studies (Chen et al., 1975; Khan et al., 1986a) provide sufficient data to permit the calculation of 95 percent binomial confidence limits for each estimate.

The Matlab data (Chen et al., 1975) show an overall U-shaped distribution: the lowest-parity women in the youngest age group and the highest-parity women in the oldest age group have the highest mortality. Women in the middle ages (20 to 39) and middle parities (2 to 5) have risks of mortality that do not seem very sensitive to increments of age or parity. While this may be an artifact of the way the data are grouped, it nevertheless suggests that younger primiparas and older grand multiparas are the women at greatest risk.

The pattern for Jamalpur (Khan et al., 1986a) is quite different, J-shaped rather than U-shaped. There is no apparent excess risk associated with a first pregnancy, which is rather surprising. This may be due to missed maternal deaths. The authors note that the crude birth rate calculated from the number of births reported in the study is 35, about 10 percent lower than the national estimated crude birth rate of 39. If births have been missed, it seems possible that maternal deaths might also have been. One social factor that would make it more likely that missed births would be first births rather than higher-order births is the custom of women going to their mother's home (and therefore away from the study area) for several months at the time of the first delivery. The increasing risk of mortality with parity in the middle age group contrasts with the rather flat pattern in Matlab.

The data for the third Bangladesh study, in Tangail (Alauddin, 1987), have been very reduced; all ages and parities have been grouped into nine cells, two of which are empty. The overall pattern is J-shaped, with very little difference apparent between 20- to 29-year-old women and those 30 or more.

The population-based study from Jamaica (Walker et al., 1985), with finer age and parity classifications than any of the studies from Bangladesh, shows the expected pattern of higher risk in the first than in the second pregnancy for all women less than 30 (Table 4). For 20 to 24 year olds and 25 to 29 year olds this pattern of decreasing risk with increasing parity continues through the fourth pregnancy, then increases sharply. For older age groups the lowest-risk pregnancy is at higher parities, suggesting that women of less than average fertility for their age may be less healthy. However, it is important to note that even at its lowest, mortality is about four times higher for these age groups than for the lowest-mortality group—the 20 to 24 year olds having their fourth child.

One hospital-based study, from 12 hospitals in Indonesia, also provides mortality ratios by age and parity (Chi et al., 1981). The level of mortality is about half that in the studies from Bangladesh, but the same U-shaped pattern is apparent.

While evidence from these five studies is suggestive rather than conclusive, it

TABLE 4 Age-parity Specific Maternal Mortality Ratios in Jamaica

Age	0	1	2	3	4–8	9	Total
< 20	0.9	0.6	–	–	–	–	0.8
20–24	0.8	0.4	0.5	0.2	1.6	–	7.1
24–29	1.4	0.7	0.4	0.3	0.7	–	9.3
30–34	–	1.0	2.4	1.6	1.2	2.1	1.8
35–39	–	5.8	6.6	3.2	8.3	1.4	2.4
40+	–	–	–	10.6	2.2	1.6	4.0
Total	0.8	0.6	0.8	0.8	1.0	1.6	1.1

Source: Walker et al. (1985).

seems that ages below 20 and above 30 enhance the simple parity-specific patterns already observed. This impression can be tested by examining several hospital series concerning primigravid/primiparous or grand multiparous women that also take age into account in describing complication rates.

Hospital Studies: Primigravid/Primiparous Women

Age-specific maternal mortality ratios for primigravidas in Zaria, Nigeria (Harrison and Rossiter, 1985) show that 20 to 24 year olds had the lowest mortality (4.8/1,000 deliveries). Below age 20, the risk of mortality varied inversely with age. Primigravidas with a stated age of less than 15 and those 15 years old had about the same very high mortality—38.5 and 41.9, respectively. Sixteen-year-old primigravidas had a mortality ratio of 14.0, and for those 17 to 19, it was 8.0. This marked pattern of higher risk with younger age is undoubtedly exaggerated by selection bias, as there is a strong local belief that home delivery is best for young girls. Primigravidas age 25 to 29 had a slightly increased risk relative to 20 to 24 year olds (6.1); those 30 or older had an even higher risk (10.1).

Among 2,291 primigravidas in Tanzania, women less than 20 years old had higher rates of anemia, eclampsia, premature labor, and mortality than women 20 to 24 years old (Table 5; Arkutu, 1978). A review of the records of women less than 20 years old who delivered at Kenyatta National Hospital in Nairobi in 1978 (Ngoka and Mati, 1980) indicated a slightly higher rate of premature delivery, with about 40 percent of the 13 to 14 year olds, 30 percent of the 15 to 16 year olds, and 20 percent of the 17 to 19 year olds delivering before 37 weeks of gestation. No mortality was observed in young primigravidas, compared with a ratio of 3.5 for the rest of the referral hospital population.

A study from Lagos (Efiong and Banjoko, 1975) that compared very young primigravidas (less than 16 years old) who had adequate or inadequate prenatal care with 22-year-old primigravidas who had adequate care indicated a higher rate of severe pre-eclampsia and eclampsia among the teenagers, even among those who had prenatal care. The authors speculate that this might be related to the nutritional status of the teenagers, who were on average from poorer families than the older primigravidas. Interestingly, the teenagers had lower rates of anemia (prenatal care, 22 percent; no care, 35 percent) than the older women (45 percent).

A similar study carried out in Jamaica from 1966 through 1969 (Hay and Boyd, 1973) found no difference in rates of pre-eclampsia or anemia but did report higher rates of premature rupture of the membranes (2.4 percent vs. 1.8 percent), antepartum hemorrhage (3.0 percent vs. 0.9 percent), prolonged labor (11.3 percent vs. 6.6 percent), and cesarean section (15.9 percent vs. 12.5 percent) *among the older women* than among the teenagers. They remark that most women, whatever their age, attend prenatal clinics regularly.

In Chile the prevalence of hypertensive syndrome increased with age at all parities, but within each age group it decreased with parity up to parity 7 (Faundes et al., 1974). Women having their first births had about double the risk of women having their second or third births at all ages except below the age of 20, when the risk for first births was three times that of second births. Primigravidas below 20 years of age were also at increased risk for hemorrhage of placental origin.

Except for the study in Jamaica, these hospital studies confirm the general pattern suggested by the population-based studies: young primigravidas are at higher risk of complications, especially pregnancy-related hypertensive disease, and of mortality than primigravidas age 20 to 24.

TABLE 5 Complication Rates and Death Rate Per 1,000 First Deliveries

Age	Anemia	Eclampsia	Pre-eclampsia	Premature Labor	Deaths	Deliveries
< 20	249	113	13	155	3.6	1,947
20–25	193	104	14	127	1.4	716
26–30	297	187	–	44	–	91
>30	162	324	–	162	–	37
All	235	116	13	144	2.9	2,791

Source: Arkutu et al. (1978).

Hospital Studies: Multigravid/Multiparous Women

In Nigeria the age of lowest risk for multigravid and grand multiparous women (5+) was again 20 to 24 years old (Harrison and Rossiter, 1985). The mortality for all multigravid women that age was 5.9 per 1,000 deliveries; for 20- to 24-year-old women of at least parity 5 it was 9.7. Risk increased with age for both groups; for all multigravid women age 30 or more mortality was 15.3, while for the grand multiparous subgroup it was 20.1.

In Chile Faundes et al. (1974) found that for hemorrhage associated with uterine inertia the major risk accrues to women of parity 5 or more, and except for women above 40 years of age this pattern is basically unaffected by age. For hemorrhage of placental origin the pattern is similar in that most women 35 or more, and most women of parity 5 or more are at greater risk. The combination of age greater than 35 and parity greater than 5 does not greatly increase risk over that for the age or parity group. As reported above, this study showed the risk of malpresentation increasing with age; at the youngest ages the risk is the same for all parities, but at all ages above 19 the risk is at least twice as high for second and third as for first births.

A study carried out in the United States in 1958–1960 reported the incidence of anemia among younger and older, white and nonwhite women of high and lower parity (Israel and Blazar, 1965). Among white women they found three times more anemia (hemoglobin, <10 grams percent) in grand multiparas (7+) than in women of lower parity. The excess was the same for women less than 35 (32 percent vs. 10 percent) as for those 35 or more (24 percent vs. 8 percent). Among nonwhite women the prevalence of anemia among grand multiparas was about the same as for white women in both age groups, but there was a much higher prevalence of anemia among lower-parity women. Thus, for nonwhite women there was at most a 20 percent excess of anemia associated with grand multiparity (32.6 percent vs. 27.4 percent for women under 34; 26.5 percent vs. 24 percent for 35+). Interestingly, in all four race-parity groups, older women were less anemic than younger women.

While these studies suggest that the risks for most complications and for death increase with both age and parity, the relationship between parity and risk is more important. Within age groups the risk of mortality or complications generally increases with parity, but within parity groups age often has no additional effect.

Short Birth Intervals

No study yet identified specifically addresses the relationship between birth-interval length and maternal mortality in developing countries. From a hospital population study in Zaria, Nigeria, Harrison and Rossiter (1985) reported that for women of the same parity the risk increases as a factor of the proportion of previous children dead. On average, women with a higher proportion of previous children dead would, in Nigeria, be expected to have shorter birth intervals; however, it is possible to postulate an exogenous factor that would account for high levels of both child and maternal mortality.

A referral hospital population study[1] carried out from 1936 to 1943 in the United States (Eastman, 1944) showed no significant difference in maternal mortality ratios by length of interval since the last live birth. The overall maternal mortality ratio was 3.1; the interval-specific ratios were 0 (0/115 deliveries) for intervals less than 12 months, 2.2 (3/1,347) for 13 to 24 months, 3.2 (7/2,191) for 25 to 48 months, and 4.0 (6/1,506) for intervals longer than 48 months. This study also provides information about some types of morbidity: there was no significant difference in the incidence of postpartum hemorrhage or puerpural fever by interval nor was any interval effect on the incidence of anemia evident. The incidence of toxemia was positively related to the length of the preceding interval (Table 6).

Because the hospital in which this study was carried out was a referral hospital and the patients are thus unrepresentative of the overall population, the possibility must be considered that this relationship is spurious. However, there is no reason to believe that the length of interval was a criterion in referral. Moreover, toxemia is but one of a number of conditions for which patients were referred, and none of the others show any relationship to interval. A more plausible alternative explanation for this relationship is the tendency for intervals to be longer for older women, in conjunction with the increased prevalence of primary hypertension with age.

Whether the prevalence of anemia increases with parity is the subject of controversy. The population-based studies of Omran and Standley et al. (1981a,

TABLE 6 Relationship Between Interval Since the Last Live Birth and Toxemia at the Time of Current Delivery

	White Ward			Nonwhite Ward			Private		
Interval	T	N	%	T	N	%	T	N	%
< 12	4	19	21.1	7	45	15.6	0	4	0
12–24	55	465	11.8	144	655	22.0	3	105	2.9
25–48	137	794	17.3	230	918	25.1	19	344	5.5
49+	139	688	20.2	140	453	30.9	19	235	8.1
Total	335	1966	17.0	521	2071	25.2	41	688	6.0

Note: T = cases of toxemia (all types); N = deliveries.

Source: Eastman (1944): Table XVII.

1981b) provide information about mean hemoglobin levels by age and parity for women from nine countries. For India and rural Turkey there is a clear inverse relationship between hemoglobin level and parity, and there is some indication of such a relationship in the data from Lebanon and Pakistan, but the information from Iran, the Philippines, Colombia, Egypt, and Syria shows no such trend.

OTHER IMPORTANT RISK FACTORS

Lack of Antenatal Care

One of the most striking correlates of maternal mortality is lack of antenatal care. The excess risk for women who have not attended an antenatal clinic can be observed by comparing maternal mortality ratios for women who attended at least once with those for women who never attended (Table 7). Except in the Gambia, women who never attended died at 2.6 to 22 times the rate of clinic attenders. While surprisingly high, the rates of antenatal clinic attendance among all patients seem representative, at least of hospitals that report this statistic in Africa and south South Asia. Among the others in the series considered for this review, reported attendance rates were 40 percent in Saudi Arabia (Chattopadhyay et al., 1986); a national rate of 85 percent in Zambia (Mhango et al.,1986); 85 percent in Harare, Zimbabwe (Frost, 1980); and 90 percent in the Kilimanjaro region of Tanzania (Armon, 1979). Reported rates of ever attendance for eight African hospitals in the maternity care monitoring program ranged from 76 percent in Bamako, Mali, to nearly 100 percent in Mwanza, Tanzania (Janowitz et al., 1984), and recent statistics compiled by Royston and Ferguson (1985) indicate rates of similar magnitude.

Hartfield (1980:71) suggests that the lower mortality among antenatal clinic attenders "is due more to a greater awareness of the benefits of hospital care and a concomitant willingness to use it than to any specific antenatal therapy." This effect would help to explain the striking reduction in mortality among even the irregular clinic attenders in rural India (Shah et al., 1971). From the Zaria data it is also clear that attendance at an antenatal clinic is strongly related to access; among women delivering in a hospital, 81 percent lived in the same town as the hospital attended, while only 17 percent of those who came from outside the town had attended (Harrison and Rossiter, 1985). Thus, when the clinic is at the hospital (as was the case in India), having had antenatal care probably proxies for living fairly close to the hospital. In Zaria it was also strongly associated with education: of those delivering in the hospital, 97.5 percent of women with any formal education had attended the antenatal clinic at least once, while only 62 percent of those without education had done so. In all but one of the eight hospitals reporting about antenatal care in the maternity care monitoring program, the median number of antenatal visits increased with the women's level of

TABLE 7 Mortality Among Patients With and Without Antenatal Care

Place	Prenatal Care No	Prenatal Care Yes	Relative Mortality[a]	Percent of All Receiving Care	Definition of Antenatal Care	Reference
Ethiopia	6.2	2.4	2.6	70	Received vs. not received; abortion deaths excluded	Kwast et al. (1987)
Gambia	23.3	23.1	1.0	73	Had an antenatal card and known to have made at least 1 visit	Greenwood et al. (1987)
Nigeria	31.2	3.9	8.0	70	Scheduled cases vs. unscheduled; 75% of scheduled delivered at home	Hartfield et al. (1980)
Nigeria	28.6	1.8	22.0	66	Booked vs. unbooked	Harrison and Rossiter (1985)
South Africa	16.3	2.0	8.2	83	One or more visits to an antenatal clinic	Barford and Parkes (1977)
Lebanon	2.0	0.2	10.0	95	Antenatal care at American University Medical Center vs. referred patients	Mashini et al. (1984)
India (urban)	15.8	1.6	9.9	83	Registered vs. not registered	Shah et al. (1971)
(rural)	8.7	0.5	(18.5)	83	Attending=regularly attending; MMR .47 in irregular attenders, 0 in regular attenders; nonattenders were treated by TBAs	Leedam (1985)
Indonesia	6.0	1.1	5.5	43	One or more visits vs. no visits	Chi et al. (1981)

[a] Calculated as maternal mortality ratio nonattenders/maternal mortality ratio regular attenders.

educational attainment (Janowitz et al., 1984). Thus, it seems likely that at least part of the beneficial effect of antenatal care is due to its correlation with access to a hospital (both physical and psychological) and with better socioeconomic status.

However, some portion of the beneficial effect occurs because antenatal clinics help prevent some complications. The advantage of regular antenatal care is that women at particular risk (e.g., because of anemia, hypertensive disease, or an obviously small pelvis) can be identified and either treated to reduce the risk or advised to deliver in a hospital. Data from Lusaka, Zambia, corroborate this effect: women who had antenatal care had considerably less risk of dying of hypertensive disease than women who did not have such care, while the rates for hemorrhage and amniotic embolism deaths are about the same in the two groups (Table 8; Mhango et al., 1986). In South Africa Melrose (1984) also reports a striking proportion of unbooked patients—83 percent—among those dying of hypertensive disease, with the proportion among puerperal sepsis deaths being about the same.

Although mortality is much lower among women who attend antenatal clinics, assessment of the quality of care at one clinic in a district hospital in Kenya (Malone, 1980) revealed deficiencies. Even after procedures were changed to facilitate identification of women at risk of complications, only 60 percent of those with abnormal conditions and 84 percent of those who required hospital delivery were correctly referred. Moreover, not all of those who are referred follow advice. One study in Nigeria reports the deaths of four women who attended antenatal clinics and who were advised to have repeat cesarean sections but who defaulted until late in obstructed labor because they wished to avoid operative delivery (Caffrey, 1979). Similar behavior has been reported from Mkar, Nigeria (Groen, 1974), and it seems likely that it occurs elsewhere in Africa.

Distance

Up to 15 percent of pregnancies will involve complications, most of which are not preventable (Rosenfield and Maine, 1985). The major factor in mortality for these cases becomes delay in arriving at the hospital. The Zaria data provide

TABLE 8 Cause-specific Mortality Ratios for Patients With and Without Prenatal Care in Lusaka, Zambia, 1982–1983

	Prenatal Care		No Care		Relative
Cause of death	N	MMR[a]	N	MMR	Risk
Hypertensive disease	5	1.2	7	9.2	7.7
Hemorrhage	9	2.1	1	1.3	0.6
Puerperal sepsis	7	1.5	2	2.6	1.7
Amniotic fluid embolism	3	0.7	0	0	–
Nonobstetric causes	8	1.9	4	5.3	2.8
All causes	32	7.4	14	18.4	2.5

[a] Maternal mortality ratio per 10,000 deliveries. Calculated assuming that the national rate of prenatal clinic attendance (85%) applies to patients at this hospital (as Mhango et al. average).

Source: Calculated from Mhango et al. (1986).

some insights into how distance affects mortality. Patients living outside Zaria had higher mortality than those from the town, but 80 percent of all deaths occurred in women who lived less than 2 kilometers from a main road. This suggests that access was a major factor in determining who got to the hospital. The excess risk was greatly reduced among women who had received antenatal care (Table 9).

This information can be used to set upper and lower limits for an estimate of the true population ratio. Since 81 percent of those living in town had antenatal care, the overall town ratio is probably lower than the true population ratio. The overall ratio for those living outside the town is probably inflated relative to the true ratio, as 83 percent of those women were emergency patients.

The pattern of people who live closer to the hospital having lower mortality is confirmed by observations in Anantapur, India, where maternal mortality in the urban area is 5.5, while in the rural area it is 8.7 (Bhatia, 1985). At all ages a much higher proportion of urban than rural deaths occurred in hospitals (Table 10).

Socioeconomic Status

In Ethiopia Kwast et al. (1986) found that when abortion deaths were excluded, illiterate women had the highest mortality (4.8 vs. 3.3); when they were included, illiterate women still suffered the most mortality, (7.1), but the rate for women with 7 or more years of education was nearly as high (6.0), while that for women with only a little education was lower (4.5; difference not statistically significant). Women from households with a monthly income of less than $25 had significantly higher mortality than women from households with a higher monthly income (15.3 vs. 3.2).

Counterintuitively, Alauddin (1987) reports for Tangail that maternal mortality is higher among women from more solvent families and among those whose families hold 2.01 to 3 acres of land (MMR = 10.3, compared with 5.3 for the

TABLE 9 The Effect of Distance and Prenatal Care on Mortality

	% from Zaria	MMR in those from:	
		Zaria	Outside
All patients	76	5.4	28.4
Booked	94	1.2	2.3
Emergency	45	23.5	34.1

Source: Calculated from data in Harrison and Rossiter (1985).

TABLE 10 Percent of Deaths Occurring in Hospital by Age and Urban/Rural Sector of Anantapur District, India

Age	Urban	Rural
15–19	91.0	22.2
20–24	73.7	12.0
25–29	25.0	22.7
30–34	75.0	13.6
35–39	60.0	25.0
40–44	66.7	0
Total	69.0	17.8
N of deaths	58	163

Source: Bhatia (1985).

women whose families hold the least land and 5.8 for the richest). Similarly, mortality is higher for those who have primary school education (14.3) than for those with no education (3.2) or secondary schooling (8.7). While some of this differential is due to higher abortion-related death rates among literate and primary-level educated women than among illiterate and higher-educated women (presumably because the rate of abortion seeking is higher in these groups), the basic pattern persists when all abortion-related deaths are excluded. Alauddin (1987) points out that the 1981–1982 Nutrition Survey of Rural Bangladesh found an inverse correlation between income and the prevalence of nutritional anemia among pregnant and lactating women and also found that women in families with intermediate-size landholdings were at highest risk of anemia.

Traditional Behavior

Birth Practices

A number of traditional birth practices contribute to higher maternal mortality, especially the use of oxytocin-containing medicines and the practice of "helping" delivery along by exerting extra abdominal pressure over the uterus.

Whether pharmaceutical or herbal, the misuse of uterine stimulants has been implicated in deaths due to obstructed labor and ruptured uterus (Armon, 1977; Rendle-Short, 1960; Elkins et al., 1985; Groen, 1974). Many of the herbal medicines used to facilitate labor in East and West Africa seem to contain ingredients with definite oxytocic action (Egwuatu, 1986). The problems arise not only from the strength of the preparations used but also the timing; all the experienced midwives interviewed in Zimbabwe said they prescribed a "delivery fast" medicine as soon as labor was established (Mutambirwa, 1985). Of additional concern is the report that many western-trained midwives practicing in both urban and rural settings considered these medicines excellent for speeding up labor and delivery, suggesting the potential for misuse of oxytocin.

Some traditional midwives also advocate the practice of bearing down starting in first stage of labor (Mutambirwa, 1985; Rendle-Short, 1960). When labor is prolonged, one of the most commonly reported ways that traditional midwives try to aid the process is by applying pressure to the abdomen, vigorously massaging the woman, squeezing the abdomen, or binding it with a cord or piece of cloth (Mutambirwa, 1985; Groen, 1974; Egwuatu, 1986). Adetoro (1987) attributes the high rate of postpartum hemorrhage observed in Nigeria to the practice of using "violent fundal pressure combined with uncontrolled cord traction" for delayed placenta.

Where intravaginal herbal medicines are used, either for obstructed labor or after delivery, the probability of sepsis is increased; this practice seems to be more prevalent in Ghana than Nigeria (Hartfield, 1980), and also exists in East Africa (Mutambirwa, 1985).

Female Circumcision

In a survey of 3,210 women age 10 or more in northern Sudan, it was found that 98.8 percent of the respondents had been circumcised, 83 percent with the most radical type of operation (El Dareer, 1983). Of these, 5 percent had to be decircumcised because of three complications—inability to pass urine, difficulty in menstruation, and difficulty in penetration—reported for 5.4 percent, 1.2 percent, and 7.3 percent, respectively, of the women. Although no study has been found that relates infibulation to maternal mortality, it is evident that the sequelae of the operation must increase the risks of childbirth. It is important to note that this practice is common only in a very small area—Sudan, Ethiopia, and Somalia (Verzin, 1975).

CAUSAL PATTERNS OF MORTALITY

To facilitate comparisons of the population- and hospital-based cause-specific reports, common categories were chosen, deaths were reclassified, and the proportion of all mortality attributed to each cause was calculated. This measure is used rather than cause-specific maternal mortality ratios mainly because of its more intuitive nature; it is easier to apprehend the meaning of a cause being responsible for 20 percent of all deaths than being responsible for 0.3/1,000 deaths, particularly when the denominator of the ratio is known to be biased, and the bias is likely to differ between hospitals. The main reason to use the cause-specific MMR would be to try to uncover a pattern of true cause-specific risk; given the quality of the data, this is extremely unlikely. The all-cause maternal mortality ratio is given for each hospital; calculation of the cause-specific ratio is very simple.

The major drawback of using proportional mortality is that overemphasis or de-emphasis of one category affects all the rest. Abortion is the category most likely to seriously affect the size of the others. Some consideration was given to the possibility of calculating proportions excluding abortion and ectopic pregnancy, so as to stabilize all other comparisons. Examination of the data suggested that the overall between-group patterns would not be significantly easier to see, and within-group proportions attributed to abortion were stable enough that little advantage would be gained.

As most authors classified deaths by main or most important cause, their judgment was considered critical in making the final determination of where to categorize a death. Care was taken to distinguish direct obstetric deaths from other deaths, but there was insufficient information to separately tally indirect obstetric and incidental deaths. Details of classification decisions are given in the appendix.

Differences by Area

Even a cursory examination of the proportional mortality for the population-based studies (Table 11) reveals some striking patterns: abortion is important in nearly every area, responsible for about 10 percent of the deaths in Jamaica, 20 percent in Bangladesh, nearly that proportion in Zambia, and nearly 30 percent in Ethiopia. Hemorrhage is a major cause of death in every study, and hypertensive disease is also important in most areas. Zambia, Ethiopia, and Gambia have high proportions of deaths attributed to infectious disease, which is responsible for very few deaths in Bangladesh, Jamaica, and India.

The hospital studies from West Africa (Table 12) show very few deaths due to abortion. Overall, hemorrhage is the most important cause, with difficult labor second, followed by infectious disease, and finally hypertensive disease. The studies from East and South Africa (Table 13) show a much larger proportion of deaths due to abortion, and hemorrhage seems less important than in West Africa. Infectious disease accounts for at least 10 percent of deaths. Hypertensive disease shows an interesting pattern, accounting for two to four times as many deaths in the series from Zambia and South Africa than in Uganda and Tanzania. Deaths from thromboembolism are also more prominent in South Africa and Zambia than in East Africa. They are virtually absent in West Africa.

Abortion accounts for about 10 percent to 20 percent of deaths in the studies from India, except for the rural area near Bombay (Table 14). Hemorrhage is again the most important single category, but hypertensive disease accounts for at least 10 percent of deaths. The proportions of deaths attributed to anemia are

TABLE 11 Proportional Mortality—Population-Based Studies

Place	Zambia	Ethiopia	Gambia	Egypt	Bangladesh			Anantapur				
	Lusaka	Addis Ababa	North Bank	Menoufia	Matlab	Matlab	Matlab	Tangail	Jamalpur	Urban	Rural	Jamaica
Year	82–83	81–83	82–83	81–83	67–68	82	76–85	82–83	82–83	84–85	84–85	81–83
Author	Mhango	Kwast	Greenwood	Fortney	Chen	Lindpaintner	Faveau	Alauddin	Khan	Bhatia	Bhati	Walker
	et al.	et al.	et al.	et al.	et al.	et al.	et al.		et al.			et al.
	(1986)	(1986)	(1987)	(1985)	(1975)	(1982)	(1988)	(1987)	(1986a)	(1985)	(1985)	(1985)
Overall MMR/1,000	1.2	4.6	23.6	1.9	7.7	5.1	5.5	5.7	6.2	5.5	8.7	1.1
N of deaths	60	45	15	437	41	39	387	48	58	40	134	193
Abortion	16.7	28.9	–	3.9	7.3	25.6	18.0	16.7	20.7	7.5	17.2	9.3
Ectopic pregnancy	6.7	–	6.7	0.7	2.4	2.6	–	–	–	–	–	10.4
Hemorrhage	10.0	6.7	33.3	28.8	12.2	17.9	19.6	37.5	17.2	17.5	23.1	19.7
Difficult labor	6.7	4.4	–	–	12.2	7.7	6.5	12.5	10.3	–	–	2.6
Sepsis	15.0	2.2	6.7	10.3	7.3	10.3	6.7	10.4	17.2	17.5	6.0	7.8
Operative	–	4.4	–	5.5	–	–	–	–	–	–	–	2.1
Hypertensive disease	20.0	6.7	6.7	5.5	24.4	15.4	11.9	14.6	20.7	17.5	18.7	25.4
Thromboembolism	5.0	2.2	6.7	0.7	2.4	2.6	–	–	–	–	–	9.3
Other direct	1.7	2.2	–	–	14.6	–	13.9	–	–	–	–	1.6
Anemia	3.3	2.2	6.7	–	4.9	–	–	4.2	–	5.0	4.5	–
Cardiovascular	1.7	–	–	16.2	–	2.6	–	–	8.6	10.0	–	2.1
Infectious	11.7	20.0	20.0	5.7	4.9	5.1	–	2.1	–	5.0	6.0	–
GI	–	–	6.7	0.9	2.4	2.6	–	2.1	–	–	–	–
Hepatic	1.7	15.6	6.7	1.4	2.4	2.6	–	–	–	5.0	6.0	–
Other	10.0	4.4	6.7	3.4	–	–	–	–	–	–	–	–
Violent	1.7	4.4	–	6.4	–	10.3	9.0	2.1	1.7	–	–	–
Other	–	15.6	13.3	16.2	7.3	–	9.0	–	3.4	20.0	24.6	5.7

TABLE 12 Proportional Mortality in Hospital Studies in Nigeria

Place	Ilesha	Ilorin	Kaduna	Kaduna	W. State	Ibadan
Year	58–70	72–83	76–77	64–72	72–73	62–71
Author	Hartfield (1980)	Adetoro (1987)	Caffrey (1979)	Balachandran (cited in Armon, 1979)	Oduntan and Odulami (1975)	Ojo and Savage (1974)
Overall MMR/1,000	12.1	4.5	6.3	–	4.3	8.2
N of deaths	175	624	141	155	212	183
Abortion	–	–	–	–	2.8	6.6
Ectopic pregnancy	1.1	–	–	–	–	3.3
Hemorrhage	19.4	24.2	9.9	25.2	30.2	15.8
Difficult labor	21.7	25.8	9.2	17.4	31.1	3.8
Sepsis	2.9	8.3	9.2	9.7	5.2	7.1
Operative	6.3	0.6	–	–	–	3.8
Hypertensive disease	12.0	11.2	7.8	17.4	10.4	3.8
Thromboembolism	0.6	0.6	–	–	–	1.6
Other direct	1.1	4.0	31.9	8.4	–	–
Anemia	6.9	7.5	–	–	9.4	18.6
Cardiovascular	0.6	–	6.4	–	–	2.2
Hemoglobinopathies	1.1	1.1	–	–	1.9	4.4
Infectious	17.1	15.1	17.0	–	–	22.4
GI	1.7	–	5.0	–	–	3.3
Hepatic	5.1	3.4	7.1	–	–	15.3
Other	10.2	11.7	5.0	–	–	3.8
Violent	–	–	–	–	–	–
Other	9.1	1.4	8.5	21.9	9.0	6.6

TABLE 13 Proportional Mortality in Hospital Studies in Africa, Excluding West Africa

Place	Tanzania Kilimanjaro	Uganda	Zambia Lusaka	S. Africa Pietermaritzburg	S. Africa Durban
Year	71–77	58–69	82–83	73–75	75–82
Author	Armon (1979)	Dept. Report (cited in Armon, 1979)	Mhango et al. (1986)	Barford and Parkes (1977)	Melrose (1984)
Overall MMR/1,000	3.3	–	1.2	4.5	1.4
N of deaths	80	69	60	118	258
Abortion	5.0	11.6	16.7	12.7	18.6
Ectopic pregnancy	5.0	–	6.7	.8	–
Hemorrhage	7.5	13.0	10.0	5.9	7.4
Difficult labor	12.5	21.7	6.7	3.4	2.3
Sepsis	8.8	15.9	15.0	22.0	11.6
Operative	–	–	–	3.4	1.6
Hypertensive disease	8.8	5.8	20.0	11.9	19.0
Thromboembolism	–	1.4	5.0	11.9	2.7

TABLE 13 Continued

Place	Tanzania Kilimanjaro	Uganda	Zambia Lusaka	S. Africa Pietermaritzburg	S. Africa Durban
Year	71–77	58–69	82–83	73–75	75–82
Author	Armon (1979)	Dept. Report (cited in Armon, 1979)	Mhango et al. (1986)	Barford and Parkes (1977)	Melrose (1984)
Other direct	13.8	2.9	1.7	–	7.0
Anemia	2.5	–	3.3	–	–
Cardiovascular	2.5	–	1.7	1.7	5.0
Hemoglobinopathies	–	–	–	–	–
Infectious	20.0	–	11.7	24.6	13.2
GI	15.0	–	–	1.7	1.6
Hepatic	–	–	1.7	21.2	8.5
Other	5.0	–	10.0	1.7	3.1
Violent	–	–	1.7	–	0.4
Other	13.8	27.5	–	1.7	11.2

TABLE 14 Proportional Mortality—Hospital Studies, India

Place	Calcutta		India	Bombay Urban	Rural	Urban	Madurai
Year	58–70	72–83	64–72	61–65	61–69	61–69	60–72
Author	Dawn et al. (1976)	Dawn et al. (1976)	Konar et al. (1980)	D'Cruz et al. (1968)	Shah et al. (1971)	Shah et al. (1971)	Rao (1975)
Overall MMR/1,000	5.9	8.4	5.6	NR	2.1	4	16.7
N of deaths	80	108	637	285	27	128	1,245
Abortion	8.8	9.3	20.9	–	3.7	14.1	8.1
Ectopic pregnancy	5.0	–	0.2	–	3.7	1.6	–
Hemorrhage	27.5	15.7	9.4	–	40.7	14.1	18.6
Difficult labor	11.3	4.6	–	15.1	–	7.0	21.0
Sepsis	3.8	8.3	3.3	9.1	14.8	5.5	5.2
Operative	3.8	2.8	3.3	–	–	1.6	–
Hypertensive disease	12.5	27.8	14.3	2.5	11.1	6.3	10.7
Thromboembolism	–	–	6.0	–	–	3.9	7.7
Other direct	–	–	3.8	–	–	–	5.0
Anemia	12.5	24.1	13.7	3.9	18.5	4.7	8.7
Cardiovascular	1.3	3.7	3.5	7.7	3.7	5.5	4.2
Infectious	10.0	1.9	15.5	20.0	–	25.0	4.8
GI	–	–	–	8.4	–	–	2.2
Hepatic	10.0	1.9	15.5	20.0	–	25.0	4.8
Other	–	–	–	9.5	–	10.2	2.2
Violent	–	–	–	–	–	–	–
Other	3.8	1.9	6.3	23.9	3.7	0.8	1.5

consistently much higher than in the East and South African studies and in most cases are higher than the proportions from West Africa. Virtually all of the infectious disease deaths are attributed to hepatitis, in contrast to the pattern in Africa, especially West Africa, and in the Middle East.

The Middle Eastern pattern (Table 15) is quite different from those in India and Africa: neither difficult labor nor hypertensive disease is very important, and anemia is totally absent. Abortion is responsible for 10 percent to 20 percent of deaths, and hemorrhage is fairly important. What is particularly striking is that the studies from Iran have larger proportions of "other infectious" deaths than any other studies, and those from Lebanon and Saudi Arabia have the highest proportions of deaths from thromboembolism.

TABLE 15 Proportional Mortality in Hospital Studies in the Middle East, Indonesia, and Vietnam

Place	Lebanon	Iran		Saudi Arabia		Indonesia	Vietnam
	Beirut	Shiraz	Shiraz	Riyadh	12 hosp	Bali	Quang Ngai
Year	71–82	63–79	70–76	78–80	77–80	81–82	67–69
Author	Mashini et al. (1984)	Daneshbod et al. (1970)	Borazjani et al. (1978)	Chatto-padhyay et al. (1986)	Chi et al. (1981)	Fortney et al. (1985)	Vennema (1975)
Overall MMR/1,000	1.3	–	2.5	.5	3.9	7.2	15.3
N of deaths	45	96	96	29	135	295	82
Abortion	4.4	8.3	7.3	10.3	NI	6.8	30.5
Ectopic pregnancy	–	–	–	–	NI	–	–
Hemorrhage	24.4	6.3	11.5	13.8	18.5	45.8	9.8
Difficult labor	4.4	3.1	–	3.4	11.9	–	11.0
Sepsis	6.7	16.7	18.8	3.4	22.2	10.5	3.7
Operative	2.2	2.1	2.1	17.2	3.7	0.3	24.4
Hypertensive disease	6.7	4.2	6.3	3.4	16.3	4.7	9.8
Thrombo-embolism	13.3	6.3	8.3	17.2	0.7	2.0	–
Other direct	4.4	5.2	8.3	6.9	2.2	–	–
Anemia	–	–	–	–	–	–	9.8
Cardiovascular	8.9	4.2	2.1	3.4	2.2	6.4	1.2
Hemoglobin-opathies	–	–	–	–	–	–	–
Infectious	6.7	36.5	19.8	13.8	0.7	8.5	–
GI	–	3.1	3.1	3.4	–	2.0	–
Hepatic	–	16.7	2.1	6.9	–	0.7	–
Other	6.7	16.7	14.6	3.4	0.7	5.8	–
Violent	2.2	1.0	5.2	–	–	2.0	–
Other direct	15.6	6.3	10.4	6.9	21.5	12.9	–

In the Indonesian studies hemorrhage and sepsis are the most important causes and possibly hypertensive disease. The high maternal mortality ratio and the pattern of important causes from the hospital in Vietnam—abortion, difficult labor, and operative—suggests that it was really a last resort.

ASPECTS OF FERTILITY AND THE MAJOR CAUSES OF DEATH

Abortion

Abortion accounts for a substantial proportion of maternal mortality in all regions covered except West Africa. The proportion of these deaths attributed by the authors to induced abortion varies. Some authors state (Walker et al., 1985) or imply (Barford and Parkes, 1977; Melrose, 1984) that all were induced. Lindpaintner et al. (1982), who made a special effort to identify induced-abortion-related deaths, report that of 10 abortion-related deaths, 1 abortion was spontaneous, 6 were reported to have been induced, and 3 were suspected to have been induced. Similar statements are common in the studies from Africa; and the study from Riyadh states that all 3 identified abortion-related deaths were assumed to be related to spontaneous abortion (Chattopadhyay et al., 1986). Thus, it is not possible to determine maternal mortality related to unwanted fertility. However, most of the evidence suggests that most abortion-related deaths are in fact due to induced abortion.

Parity-specific patterns are very different in Africa and Asia. The population-based studies from Bangladesh and hospital studies from India suggest that in Asia most deaths are to multiparous women. This pattern is confirmed by a population-based study of abortion in Jamalpur, Bangladesh, which found that the induced-abortion/live birth case ratio increased from 32/100 for primiparous women to 37 for parities 1 to 4 and 96 for women of parity 5 or more (Khan et al., 1986b). Moreover, case fatality also increased with parity, from about 1 percent for primigravidas to 3 percent for those of parity 5 or more. Most induced abortions were obtained by married women. Only 12 percent were obtained during the second month of pregnancy and 49 percent during the third.

A hospital-based study of women admitted for complications of induced abortion in Dhaka confirmed the high parity and married status of most women who obtain an abortion in Bangladesh (Khan et al., 1984). The hospital case-fatality rate was 7.7 percent; and compared with women who survived, the women who died were less likely to be married (55 percent vs. 91 percent), more likely to be slum dwellers (58 percent vs. 23 percent), more likely to have gone to a nonmedical practitioner (95 percent vs. 45 percent), and more likely to have had abortion induced by insertion of solid objects into the uterus (71 percent vs. 30 percent). The major complications were uterine perforation, excess bleeding, and sepsis.

Interviews with health workers throughout Bangladesh about pregnancy-related deaths in 1978-1979 revealed that an average of 25 percent of identified deaths were due to induced abortions, with estimates for specific districts ranging from 16 percent to 48 percent (Rochat et al., 1981). The estimated proportion of maternal deaths due to abortion in Tangail District was 33 percent, about twice the 17 percent obtained from the population-based study carried out 5 years later (Alauddin, 1987). The health worker interviews suggested a proportional mortality of 21 percent in Mymensingh District, the site of the Jamalpur population-based study, which also obtained a proportional mortality of 21 percent (Khan et al., 1986a).

The population-based study in Anantapur India (Bhatia, 1985) and the hospital-based studies from India suggest that proportional mortality of abortion is at about the same level or slightly lower than in Bangladesh. One hospital-based study of abortion in Thailand (Chaturachinda et al., 1981) suggested that women who have abortions there, like those in Bangladesh, tended to be older and to have more children. A survey of knowledge, attitude, and practice in one region of the Philippines in 1967–1969 also showed a similar pattern and found that the prevalence of ever having had an induced abortion was 30 percent (Flavier and Chen, 1980).

In contrast, 12 percent of Turkish women with only one or two living children said in response to a direct question that they had had an induced abortion; the proportion estimated by randomized response technique in a similar population was 30 percent for those with one or two children and 17 percent for those with none (Tezcan and Omran, 1981).

The pattern in Africa is similar to that in Turkey: Aggarwal and Mati (1980) found that 53 percent of women hospitalized with septic abortion in 1978 in Nairobi were less than 20 years old, compared with 35 percent of those with nonseptic abortions. Infection is used as a proxy for induced abortion, as women do not necessarily admit to having induced an abortion. Aggarwal and Mati state that most patients were primigravidas. Eighty-three percent were in their second trimester, and the mortality ratio was 2/1,000 abortion admissions (12.5/1,000 septic abortion admissions).

A study of 59 women in Nigeria who were admitted for complications of abortion and admitted to having induced the abortion showed that 52 percent were less than 18 years old (Okojie, 1976). For 81 percent it was the first pregnancy; most were single, and 58 percent were secondary school students, suggesting that abortion was being used to delay a first birth. Other studies from Nigeria support this conclusion (Unuigbe et al., 1988; Adetoro, 1989). This was also the conclusion of Lamptey et al. (1985), who surveyed women coming for delivery in a hospital in Accra, Ghana. They found that 25 percent reported having at least one induced abortion, and they mention that the case-fatality ratio in their hospital for induced abortions was at least 34 per 1,000.

Given this reported prevalence and extremely high case-fatality rate, it is surprising that abortion figures so little in the hospital-based studies of maternal mortality from this area. One possibility is that deaths are missed because women are admitted to a different section of the hospital; both the Aggarwal and Mati (1980) and the Okojie (1976) papers discuss induced or suspected induced abortions solely in the context of gynecologic emergency admissions. A second possibility is that behavior in the capital city—Nairobi or Accra—is different from that in more provincial cities and that abortion rates are higher. If the reported reluctance of women to admit having recently induced an abortion reflects a reluctance to admit it implicitly by seeking care, inclusion bias probably plays a stronger role in exaggerating case-fatality rates for abortion than for delivery mortality. Nevertheless, induced abortion resulting from unwanted fertility clearly increases maternal mortality.

The pattern of utilization of abortion by young primiparous women implies that it may be difficult to reduce maternal mortality from this cause by increasing contraceptive use. The chances for that are much greater in Asia, where not only are the women who seek abortion older but also, at least according to one series of interviews with abortion providers, their husbands agree with their decision (Islam, 1982).

Difficult Labor

Difficult labor is more important as a cause of maternal mortality in West Africa than anywhere else, although the studies from East Africa also show high proportions. A large number of these deaths are associated with uterine rupture.

A number of case series studies concern uterine rupture (Table 16). Incidence rates range from 0.7 to 10.8 per 1,000, no doubt at least partially due to variation in the prevalence of hospital delivery. What is most striking is the very high associated mortality: the lowest among the series is 7 percent and the highest is 42 percent, with a median rate for all studies of 10 percent. Much of this excess mortality is due to late arrival at the hospital; except for the series from South Africa, fewer than 20 percent of patients in any study had had any antenatal care, indicating that they arrived as emergency admissions.

The distribution of type of rupture differed widely between studies. From 1 percent to 63 percent were attributed to rupture of a scar, usually a previous cesarean section, but occasionally a repaired rupture or tear and in a few cases sequelae of an induced abortion. Between 6 percent and 32 percent were attributed to trauma, mostly arising from manipulations during attempts to correct malpresentation. In all series except the one from Cameroon, most ruptures were considered to be spontaneous. More than 50 percent of these were associated with fetopelvic disproportion, in several cases exacerbated by traditional attempts to remedy the situation (use of oxytocin-containing medicine, abdominal pressure on uterus).

These hospital series of uterine rupture contain few primigravidas. The average parity for nearly all series was about 4, and risk apparently increases with

TABLE 16 Uterine Rupture in Africa: Incidence, Case-Fatality, and Associated Risk Factors

Country	Year	Deliveries	Cases /1,000	Case–Fatality	Percent due to Scar	Trauma	Spontaneous	% Parity < 5	% Unbooked	Author
Cameroon	1973–76	36,977	1.9	8.5	63	32	5	50	NR	Nasah and Drouin (1978)
Nigeria	1965–72	16,189	8.7	7.8	1	6	93	42	NR	Groen (1974)
Nigeria	1974–80	15,872	6.7	7.0	22	10	68	NR	4	Megafu (1985)
Nigeria	1978–83	6,000	10.8	20.0	24	13	63	NR	16	Elkins et al. (1985)
S. Africa	1966–74	182,707	1.8	42.0	16	12	63	83	3	Mokgokong and Marivate (1976)
S. Africa	1973–77	126,713	.7	9.7	34	2		76	77	Golan et al. (1980)
Malawi	1967–70	5,030	8.9	37.8	25	21	54	52	NR	Armon (1977)
Tanzania	1971–76	17,300	4.0	8.6	25	21	54	52	NR	Armon (1977)
Uganda	1952–58	15,908	10.7	36.8	22	14	64	74	20	Rendle-Short (1960)

parity. However, the proportion of women with rupture who are at less than parity 5 ranges from 42 percent to 83 percent (Table 16).

In a retrospective study of maternal height as a predictor of disproportion in primigravidas in Sierra Leone, almost 8 percent of a consecutive series of clinic attenders had cesarean section for disproportion (Aitken and Walls, 1986). While one criterion for inclusion in the study was a complete record, including height and weight, few records were rejected for incomplete data, and there is no suggestion that height was selectively recorded for those thought to be at risk of disproportion. If this is taken to be a representative population, the implication is that cesarean section averts some of the deaths that would otherwise occur because of disproportion and uterine rupture. Five of the hospital studies of maternal mortality include information about cesarean section rates and about associated case fatality (Table 17). Unfortunately, none of them report parity-specific rates or give much detail about the reasons for the cesareans, so it is difficult to assess the fertility-associated risk of disproportion. Aitken and Walis (1986) state that it is highest for primigravidas, but they offer no evidence.

Studies in Latin American hospitals in Honduras, Venezuela, Chile, and Brazil show a much higher incidence of cesarean section, ranging from 5 percent in Honduras to 41 percent in Brazil (Janowitz et al., 1982a). Associated case-fatality rates ranged from 0 (Honduras and Brazil) to 0.3 percent (Venezuela); maternal mortality per 1,000 vaginal deliveries in the same hospitals ranged from 0 (Brazil) to 0.3 (Venezuela), much lower than in any of the hospital series from Africa or Asia. The age-parity-specific incidence rates showed the same pattern in all four hospitals: highest in primigravidas and decreasing with parity. Age above 30 years increased the risk of having a cesarean section at all parities but especially for primigravidas, with this multiplicative effect decreasing with parity. Across all hospitals the major recorded indication for primigravidas having a cesarean section was obstructed labor, but from 27 percent to 83 percent of cases had no recorded indication. A second study, of two hospitals in Brazil, suggested that recorded indications are not a good criterion of true risk; incidence of cesarean was most strongly related to method of payment (Janowitz et al., 1982b).

TABLE 17 Incidence and Case-fatality of Cesarean Sections

Country	Cesareans /1,000 deliveries	% Case-Fatality	Reference
India	13.5	2.3	Rao (1975)
Vietnam	39.4	8.5	Vennema (1975)
Saudi Arabia	2.8	0.3	Chattopadhyay et al. (1986)
Tanzania	52.3	0.8	Armon (1977)
Indonesia	78.0	1.6	Chi et al. (1981)

Even though the underlying causes for the observed inverse relationship of parity and risk of cesarean section cannot be precisely determined, the results of these studies point in the same direction as the information from the series about uterine rupture: a great deal of the risk of death from difficult labor occurs to women of lower parities. The potential for averting these deaths through reducing fertility may be limited.

Hemorrhage/Anemia

Hemorrhage is the most consistently important cause of death across all areas covered by the hospital studies. It is associated with a large number of conditions, including obstructed labor and placental pathology, which are not very susceptible to simple methods of detection or prevention. Late arrival at hospital is a major cofactor in hemorrhage deaths (Armon, 1979; Chi et al., 1981).

Although underlying anemia can be a cofactor in death from hemorrhage, observed associations between anemia and maternal mortality probably more often reflect the effect of hemorrhage and delay in hospitalization. Anemia measured from blood taken at admission reflects blood loss due to the complication as well as underlying anemia. Women who have lost more blood before arriving at the hospital are probably more likely to die; thus the relative risk of mortality associated with anemia is probably overestimated, and many "anemia" deaths are likely to be mainly due to hemorrhage.

Underlying anemia can exacerbate the effects of hemorrhage: with the same amount of blood loss, anemic women suffer from shock more quickly than nonanemic women (McFee, 1973). Most underlying anemia is due to nutritional deficiencies; usually lack of iron or folic acid. Some is due to malaria infection (see infectious disease section below) and a small amount is a consequence of hemoglobin SS or S-C disease.[5] Anemia is most prevalent in Asia, particularly South Asia; followed by West Africa, then East and South Africa; with Latin America having the lowest rates (Table 18), and is more common in rural areas than in cities (Royston, 1982). It is strongly associated with poverty. In addition to contributing to deaths from hemorrhage, anemia is an important factor in cardiac failure.

In the hospital series reviewed, anemia is cited as a factor in many of the deaths in Bangladesh (Alauddin, 1987), Tanzania (Armon, 1979) and Nigeria (Ojo and Savage, 1974), where it is noted as being most severe in primiparas. Chattopadhyay et al. (1986) report that the prevalence among the obstetric population in Riyadh is less than 1 percent. Only two studies cite hospital population preva-

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[5] These are really significant only in West Africa, where they occur at low prevalences in the reproductive population. In fact, sickle cell disease (Hb SS), which carries the greatest risk of death—50 percent in one series—is a rarer complication than Hb SC disease, because so few who have it survive to reproductive age (Harrison, 1976).

TABLE 18 Estimated Percentage of Pregnant Women With Hemoglobin < 10 g/100 ml.

Africa	
North	
Algeria	65
Egypt	75
Libya	47
Morocco	46
Tunisia	38
West	
Gambia	80
Ghana	64
Guinea-Bissau	85
Ivory Coast	34
Mali	50
Mauritania	24
Niger	24
Nigeria	65
Sierra Leone	45
Togo	47
East	
Ethiopia	6
Kenya	48
Malawi	49
Mauritius	80
Uganda	35
Tanzania	59
Zambia	60
Zimbabwe	27
South	
South Africa	25
Latin America	
Central America	
Costa Rica	44
El Salvador	15
Guatemala	34
Mexico	38
Nicaragua	20
Tropical	
Brazil	20
Colombia	22
Guyana	55
Peru	35
Venezuela	52
Temperate	
Argentina	61
Chile	32
Oceania	
Fiji	68
Papua New Guinea	55
Asia	
West	
Iran	50
Israel	29
Lebanon	50
Turkey	74
South	
Bangladesh	66
India	68
Nepal	33
Pakistan	65
Sri Lanka	62
Southeast	
Burma	55
Indonesia	65
Laos	62
Malaysia	77
Philippines	47
Singapore	26
Thailand	48
Vietnam	50

Source: Royston (1982).

lence and give case-fatality rates. In Madurai, India, the prevalence of severe anemia (< 7 grams percent) was 3.2 percent among all women coming for delivery; the maternal mortality ratio of severely anemic women was 45.2, compared with 15.8 for the rest of the population (Rao, 1975). In Indonesia, the prevalence of anemia (< 10 grams percent, measured on admission) was 47.7 percent for rural and 31.4 percent for urban women (Chi et al., 1981). The maternal mortality ratio was 13.5 for rural anemic women but only 2.3 for nonanemic women. For urban women the differential was less: 3.8 for anemic, compared with 1.8 for nonanemic women. The authors suggest that because of transportation difficulties rural women arrive later during complicated deliveries than urban women do and have thus suffered a greater loss of blood prior to admission.

In the hospital studies reviewed that discuss association with age or parity, hemorrhage is remarked to be a cause of death associated with high parity and older age: 75 percent of the hemorrhage deaths in Durban were to women more than 25 years old (Melrose, 1984), while in Pietermaritzburg most were above the age of 30 and of parity 5 or more (Barford and Parkes, 1977). In Jamaica the maternal mortality ratios for hemorrhage increase with age up to 8.1 for those 35 to 39 and 5.1 for those 40+ (Walker et al., 1985). In Chile most of the risk of hemorrhage, whether due to placental pathology or uterine inertia, accrued to women of parity 5 or more, except that primigravidas less than 20 were also at higher risk of placental hemorrhage (Faundes et al., 1974).

Thus, except for malaria-associated anemia (see below), any increase in risk of hemorrage death because of the prevalence of anemia is either stable or increases with parity. Moreover, there is evidence that hemorrhage occurs mostly in older multigravid women. Hemorrhage deaths, then, are one category of maternal mortality that might be reduced by a reduction in fertility.

Hypertensive Disease of Pregnancy

Virtually all of the studies reviewed, whether population based or hospital based, and whether concerned with mortality or morbidity indicated that hypertensive disease of pregnancy is most common among primigravidas. The pattern of increased risk for younger primigravidas below the age of 20 may reflect not so much increased physiologic risk as other differences between them and women who have their first child at age 20 to 24. In a recent prospective study of primigravidas in Vietnam, Burma, Thailand, and China (WHO International Collaborative Study, 1988), clinical diagnosis of hypertensive disorder varied greatly, from about 1 percent of the women in Vietnam and Thailand, to 5 percent of those in Burma and 31 percent of those in China. The proportions of women having different signs of hypertensive disease differed among the countries, but for all signs except proteinuria the proportion was highest in China and lowest in Vietnam. The most striking finding was that mean diastolic blood pressure was remarkably constant across populations through the second trimester; in the third trimester there was little increase in Vietnam or Burma, there were greater increases in Thailand, and the largest increase was in China. Whether due to genetic differences, as the authors suggest, or to subtle differences in behavior, it is clear that cross-national differences are significant.

Hypertensive disease noticed in older primigravidas and in a few studies among older grand multiparous women (e.g., Barford and Parkes, 1977) may reflect the increased risk of essential hypertension with age.

Infectious Disease

Although not many studies deal explicitly with this aspect of maternal mortality, it is clear that pregnancy increases the case-fatality rate of certain infectious diseases—infectious hepatitis, malaria in some circumstances, and possibly others—probably through alterations in the immune system in response to pregnancy. In tetanus-endemic areas, abortion and delivery offer excellent opportunities for the organism to infect women.

Hepatitis

In the population and hospital studies reviewed, infectious hepatitis is the single most important disease in terms of increasing the risk of maternal mortality. It is not clear whether pregnant women are more susceptible to hepatitis than nonpregnant women, but they are at greater risk of death from the disease than nonpregnant women. Case series reports demonstrate case-fatality rates that range from 10 percent to 61 percent (Table 19), much higher than for the general population. The series that deal exclusively with pregnant women have been criticized on the grounds that these rates might be spuriously high because only seriously ill women would be admitted to a hospital. Several reports compare hospital case-fatality rates for pregnant women with those for nonpregnant women and men; these confirm that pregnant women are at particular risk and also that women in general are at greater risk than men (Table 19).

There remain several questions concerning possible selection bias. Christie et al. (1976) considered whether pregnant women with jaundice might be more likely to be admitted than nonpregnant women with jaundice. This possibility was rejected; however, it is important to note that even if such a bias existed, it would tend to reduce the case-fatality rate among pregnant women. A more serious problem is whether women come later and are more severely ill on admission than men and whether this tendency is greater for pregnant than for nonpregnant women. Gelpi (1978–1979) rejected the first part of this hypothesis, finding no significant difference by sex in the proportion of fatal cases presenting in coma. In Ghana, Morrow et al. (1968) found no substantial difference in hospitalization rates by sex for patients from the Accra Municipal Council Area

TABLE 19 Case-fatality Rates per 100 Hospitalized Patients With Hepatitis

Place	Year	Male	Female Nonpregnant	Pregnant	Reference
India	1949	7.8	27.3	50.5	Wahi and Arora (1953)
Saudi Arabia	53–63	4.5	11.9	46.3	Gelpi (1978)
Libya	1975	.5	1.6	13.0	Christie et al. (1976)
Bombay	65–66	–	25.6	53.8	D'Cruz and Balani (1968)
South Iran	55–70	–	18.0	44.0	Borhanmesh et al. (1973)
South Iran	67–70	–	–	26.0	"
Algiers	1956	–	–	61.1	Haemmerli (1966)
India	1956	–	–	44.8	"
Haifa	1959	–	–	30.0	"
Athens	1962	–	–	25.9	"
Jerusalem	1947	–	–	18.5	"
Cordoba	1957	–	–	16.3	"
Ghana	62–63	–	–	13.3	Morrow et al. (1968)
Haifa	50–57	–	–	9.2	Peretz et al. (1959)

(for males, 30.9 per 100,000; for females, 34.8), but pregnant women had higher case-fatality rates and a higher risk of serious disease.

Haemmerli (1966) raised the question of genetic susceptibility, noting the predominance of high case-fatality rates in the circum-Mediterranean area; this seems unlikely given the additional case series reports from India, Ghana, and Ethiopia (Wahi and Arora, 1953; Morrow et al., 1968; Kwast and Stevens, 1987) as well as the high proportional mortality from hepatitis reported in some of the hospital maternal mortality series (D'Cruz and Balani, 1968b; Shah et al., 1971; Konar et al., 1980; Barford and Parkes, 1977; Ojo and Savage, 1974).

The generally higher case-fatality rate for nonpregnant women than for men has been attributed to the lower nutritional status of women, but no evidence has been offered to support this.

Tuberculosis

Whether women with untreated tuberculosis are at higher risk of maternal mortality is unclear. Although this was the prevailing opinion in Europe and North America beginning in the late 19th century through the 1940s and currently prevails in many high-mortality countries today, it is possible that pregnant women with tuberculosis really die at no greater rate than nonpregnant women with tuberculosis. No study from a high-mortality country that addresses this question has been identified. However, a careful review by Hedvall (of European studies) in 1953 showed about as many that demonstrate no adverse effect or even a favorable effect of pregnancy on tuberculosis as those that show a negative effect. In Hedvall's (1953) own prospective study of pregnant women with pulmonary tuberculosis, 9 percent improved during pregnancy and 7 percent worsened.

Women who are adequately treated probably have no higher risk. In a comparison of pregnant and nonpregnant tubercular women, with both groups having similar severity of disease and being treated similarly for active tuberculosis, Flanagan and Hensler (1959) showed no difference in disease progression.

Malaria

During an epidemic of malaria in Ceylon the case fatality rate was twice as high among pregnant (13.1 percent) as among nonpregnant (6.5 percent) women (Wickramasuriya, cited in McGregor, 1986). This pattern of enhanced risk of mortality from *P. falciparum* during pregnancy is probably generalizable to areas of unstable (epidemic) transmission (Brabin, 1983). In hyperendemic areas where immunity to the parasite is high because of repeat exposure, pregnancy does not seem to have the same amplifying effect. However, a number of studies have shown that in these stable-transmission areas malaria parasitemia is more frequent and heavier during pregnancy, and particularly during the first and to a lesser extent during the second pregnancy (McGregor et al., 1983; McGregor, 1986). One direct consequence of this for maternal health is an increased probability of anemia (Gilles et al., 1969; McGregor, 1986; Brabin, 1983).

CONCLUSION

The basic pattern observed for the relationship between fertility and maternal mortality is that risk of mortality is highest for first pregnancies and for fifth and subsequent ones. This pattern exists whatever the overall level of maternal mortality. Extremes of age, lack of medical care, poverty, and some infectious disease cofactors increase the risk. Thus, the lowest age-parity-specific risk in Jamaica is only half to a third as large as that in Bangladesh or Indonesia. These conditions also increase the relative risk of first and high-order pregnancies: the risk of first pregnancies relative to middle pregnancies is higher in Asia and Africa than in Jamaica. As conditions improve, the U-shaped curve of risk with parity is not only lower but also flatter.

Given this pattern the potential for fertility reduction alone affecting maternal mortality is limited. The possible mechanisms through which fertility reduction can occur are contraception and provision of safe induced abortions. For first births use of contraception or safe abortion could reduce risk of mortality either through allowing postponement of the first birth until after age 20 or through averting unwanted births. Even if contraception is available, use by teenagers may not be high, so provision of safe abortion is probably the most effective way to reduce mortality associated with unwanted first pregnancies. However, in many countries there are severe constraints to providing either contraceptives or abortions to young unmarried women, who are the most likely to have unwanted first pregnancies.

The potential is greater for reducing mortality associated with unwanted higher-parity births. Family planning programs typically affect fertility mainly through reducing the number of high-parity births. Thus, except in sub-Saharan Africa, substitution of safe for unsafe abortions and contraception for abortion could substantially reduce maternal mortality—20 percent to 25 percent of all maternal deaths in some parts of Asia are associated with abortion. However, this effect will be limited by the effectiveness of programs to convince women to use contraception.

As for the middle parities, there is as yet insufficient evidence about whether the length of the interval between births has any effect on risk of maternal mortality.

It is likely that maternal mortality ratios—the risk of death per pregnancy—will increase, at least in the short term, in some areas as family size decreases. This paradoxical effect arises because of the way family planning programs typically affect fertility. First, as the number of high-parity births decreases, high-risk first births form a larger proportion of all births. Second, there is strong evidence for a population of at least two segments, one of which is in contact with the official health system, gets prenatal care, and has risks that are well below those of the other segment. It is likely that women who elect to contracept to postpone early childbearing, to end childbearing early, or to increase intervals between births will initially be those at lower risk of maternal mortality. Thus, reducing the high-parity fertility of these women may exclude those who have higher intrinsic risks. It is important to remember, however, that even when the risk per birth increases, the absolute number of deaths to women will probably decrease.

Examination of the causal patterns of mortality suggests some alternative routes to reduce maternal mortality. For all causes whatever is measured by "antenatal care" is important. The Zaria data show that it is not simply physical access to a hospital, though that is clearly important. It would be helpful to know for those living close to a hospital if having been to an antenatal clinic reduced the interval between development of a complication during home delivery and going to the hospital. It is unfortunate that the population-based studies have not paid more attention to antenatal care and hospital attendance for complications as factors in mortality.

APPENDIX—CONTENTS OF CAUSAL CATEGORIES

The abortion category includes all abortion-associated deaths, whether induced or spontaneous, because many studies either do not mention whether abortion was induced or indicate that it was determined indirectly. Hemorrhage includes all deaths attributed to hemorrhage, antepartum hemorrhage, placenta previa, placental abruption, postpartum hemorrhage, and retained placenta that were categorized by the author as hemorrhage rather than sepsis. A few deaths attributed to hemorrhage and ruptured uterus were classified under difficult labor, along with deaths attributed to ruptured uterus (whether or not there was a previous scar) and obstructed labor, including deaths due to disproportion and malpresentation. The sepsis category includes deaths attributed to puerperal tetanus and septicemia. If the author classified a death as due to cesarean section, anesthesia, sepsis, or hemorrhage connected with cesarean section or to anesthesia, the death was classified as operative, even if it was highly probable that the section was performed for obstruction or abruption. It is clear that the categories of hemorrhage, difficult labor, sepsis, and operative overlap, in that many deaths could almost as easily be put in one category as in another.

Hypertensive disease includes deaths due to toxemia and eclampsia. Under thromboembolism are grouped deaths ascribed to pulmonary, amniotic fluid, and air embolisms as well as those due to cerebrovascular hemorrhage. This last category probably includes a number of deaths with toxemia as an underlying cause.

The cardiovascular category includes mainly deaths attributed to congenital heart disease (mitral stenosis) and rheumatic heart disease or simply to cardiovascular disease. It is not clear how frequently cardiac failure due to anemia is classified as cardiovascular. The gastrointestinal infectious category includes amebiasis, typhoid, and cholera deaths. All deaths attributed to hepatitis, infectious hepatitis, or hepatic coma when it is clear that there was an epidemic (either because the author says so or because the proportion of deaths so indicates) were attributed to hepatitis. Isolated hepatic or jaundice deaths that were not called infectious hepatitis are classified as "other indirect." The "other infectious" category includes mainly incidental deaths: anthrax (from Iran), smallpox, meningitis, tuberculosis, malaria, and pneumonia. Suicide, murder, and one motor vehicle death make up the "violent death" category. The "other" category includes deaths due to epilepsy and various cancers, most of which seem to be incidental.

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Health Effects of Contraception

Nancy C. Lee, Herbert B. Peterson, and Susan Y. Chu

INTRODUCTION

Until the 1960s rhythm and barrier contraceptives were the only methods of birth control widely available to couples desiring to plan the number and spacing of their children. In the 1960s oral contraceptives (OCs) were introduced and new efficacious intrauterine devices (IUDs) became widely available, so that the choice of effective methods of contraception increased substantially. Later, in the 1970s, female and male sterilization techniques became much more widely accepted and used. Couples were then able to choose from several different temporary and permanent methods of contraception and to switch from one to another. Worldwide, family planning programs expanded, and the prevalence of contraceptive use increased.

As these methods of contraception became more widely used, anecdotal reports of adverse health effects associated with their use began to appear. Since the late 1960s and early 1970s, epidemiologic studies have more rigorously evaluated the health effects associated with the use of different contraceptive methods. Most of these studies have been conducted in the United States and Europe. In the process researchers have recognized that different contraceptive methods have

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important beneficial health effects, in addition to the desired effect of preventing pregnancy. Although much research is still needed, especially targeted to the developing world, a large body of information is now available to assess the health effects of the various contraceptive methods.

The various contraceptive methods have health risks, but pregnancy itself has attendant risks of morbidity and mortality. In 1983, Ory et al. attempted to quantify the mortality risks associated with using the various methods of contraception. Those risks were compared with the risks associated with using no method of contraception, which are actually the mortality risk associated with pregnancy. These estimates are presented in Table 1. Using no method of contraception carries a higher cumulative risk of death than using any contraceptive method except that of OCs by older women who smoke. The mortality risks associated with using no contraception and with using OCs are higher in older women than in younger women. For all other contraceptive methods, the mortality risk does not appear to vary by age. Although the estimates are not presented in Table 1, vasectomy involves no mortality risk for women and virtually none for the male partner (Ory et al., 1983).

Below we will present in detail the health effects of the various widely available methods of contraception, limiting our discussion to those methods that are considered moderately to highly effective. Most epidemiologic and clinical studies of the health effects of contraceptives have been carried out in developed countries. We recognize the difficulty in generalizing these results to the special health and cultural situations in the less developed countries. Furthermore, the

TABLE 1 Estimated Cumulative Number of Deaths per 100,000 Nonsterile Women Aged 17-44, Attributable to Contraceptive Method, by Age Group

Method	Age Group 17-34	35-44	Total (17-44)
No method	179	270	449
Pill/nonsmoker	20	230	250
Pill/smoker	127	845	972
IUD	22	20	42
Condom	17	4	21
Diaphragm/spermicide	24	25	49
Rhythm	31	32	63
Tubal sterilization[a]	—	—	4

[a] Not available by age group.

Source: Modified from Ory et al., 1983, p. 36, Figure 16.

effects of the various contraceptive methods on the risk of diseases generally limited to less developed countries have had very little characterization. Because of the expanding role of family planning programs and contraceptive use in these countries, studies to evaluate the health effects of contraceptives in various regions and cultures are needed.

Modern contraceptive methods vary substantially in how effectively they prevent pregnancies. Because pregnancy itself has attendant health risks and benefits, the rates of accidental pregnancy associated with the various methods of contraception are one important aspect to consider when measuring the health effects of these methods. In 1987 Trussell and Kost published their comprehensive review assessing failure rates for each method of contraception. After reviewing all available studies, they estimated the rate of failure (i.e., accidental pregnancy) in the first year of use associated with "perfect" use of each contraceptive. They called this estimate the "lowest expected" failure rate; this rate should reflect the frequency of failures caused by the contraceptive itself. They also reported for each method a "typical" failure rate, defined as the rate of accidental pregnancy in the first year of use among typical couples who use that method. The typical failure rate is determined both by failures as a result of imperfect use of a contraceptive and by failures directly related to the method itself. Most of the typical failure rates were derived from national surveys of U.S. women. The authors summarized these rates, by contraceptive method, in a single table (Trussell and Kost, 1987, p. 271). We present a modified version of that table here (Table 2). In our table and throughout the text we have substituted the term *method failure rate* for *lowest expected failure rate* and *user failure rate* for *typical failure rate* to follow more closely the terminology used in much of the existing literature. Note that the method failure rates are consistently low for most modern contraceptives. However, user failure rates vary widely, a function of the degree of acceptability and compliance required for successful use of each method.

ORAL CONTRACEPTIVES

OCs, a highly effective method of birth control, are available in two types. Combination OCs, the most widely used, consist of both an estrogen and a progestin component. Most combination OCs contain a fixed daily dose of an estrogen and progestin and are taken for 21 of 28 days of each menstrual cycle (Hatcher et al., 1988). Recently introduced, phasic combination OCs contain varying doses of the estrogen and progestin components throughout the menstrual cycle. The second type of OC is the progestin-only pill (often called the minipill), which contains only a progestin. Combination OCs with fixed doses of estrogen and progestin have been used much more frequently than phasic or progestin-only pills; hence, most epidemiologic studies on the health effects of OCs are essentially studies of the effects of this type of OC.

TABLE 2 Method Failure and User Failure Rates During the First Year of Use of a Contraceptive Method, United States

Contraceptive Method	Percentage of Women Experiencing an Accidental Pregnancy in the First Year of Use	
	Method Failure[a]	User Failure[b]
Pill	3	
Combination	0.1	
Progestin-only	0.5	
IUD		6
Medicated	1	
Nonmedicated	2	
Injectable progestin		
DMPA	0.3	0.3
NET	0.4	0.4
Implants (NORPLANT®)	0.2	0.2
Diaphragm with spermicide	3	18
Condom	2	12
Spermicides[c]	3	21
Sponge (Today™)		
Nulliparous	5	18
Parous	>8	>28
Cap/spermicide	5	18
Periodic abstinence		20
Ovulation method	8	
Symptothermal	6	
Calendar	10	
Postovulation	2	
Withdrawal	4	18
Female sterilization	0.2	0.4
Male sterilization	0.1	0.15

[a] Among couples who use a method perfectly (both consistently and correctly), an estimate of the percentage expected to experience an accidental pregnancy during the first year if they do not stop use for any reason.

[b] Among typical couples who use a method, the percentage who experience an accidental pregnancy during the first year if they do not stop use for any reason. Includes failures due to the method itself as well as failures due to imperfect use of the method.

[c] Foams, creams, jellies, and vaginal suppositories.

Source: Modified from Trussell and Kost, 1987, p. 271.

OCs prevent pregnancy chiefly by inhibiting ovulation in almost all menstrual cycles, although OC-related changes in the cervical mucus and endometrium may also have contraceptive effects. Failure rates associated with OC use are low. The method failure rate for combination OCs is 0.1 percent per year and for progestin-only OCs is 0.5 percent per year (Table 2). The user failure rate cannot be readily determined for the two types of OCs separately, but it is about 3 percent per year for any type of OC. Because this user failure rate was derived from data where the vast majority of women used combination OCs, the figure probably is closest to the user failure rate for combination OCs. Most experts believe that the progestin-only pill has a higher user failure rate.

The health risks and benefits of OC use have been extensively studied and documented (Ory, 1982; Ory et al., 1983; Stadel, 1986; Prentice and Thomas, 1987). For a recent extensive review and list of references, see Stadel (1986) or Prentice and Thomas (1987).

Because OCs are highly effective at preventing any pregnancy, they appear to decrease greatly the risk of ectopic pregnancy. Results from a large case-control study of ectopic pregnancies conducted in the United States showed that current OC users had a relative risk of ectopic pregnancy of 0.1 (95 percent confidence interval, 0.1–0.2) compared with women who were using no contraceptive method (Ory, 1981).

Noncontraceptive Benefits

An important benefit from OC use is a reduction in risk of two serious reproductive system cancers, endometrial and ovarian cancers. This reduction has been documented in at least 9 and 11 epidemiologic studies, respectively (The Cancer and Steroid Hormone Study [CASH], 1987a, 1987b). Although the theoretical mechanisms that may explain these protective effects are quite different for the two types of cancer, the magnitude and characteristics of the protective effects are similar. The most detailed characterization of these protective effects comes from the CASH Study, a large case-control study conducted in the United States by the Centers for Disease Control, with support from the National Institute of Child Health and Human Development (CASH, 1987a, 1987b). OC use was associated with a 40 percent reduction in the risk of endometrial cancer as well as a 40 percent reduction in the risk of ovarian cancer, regardless of the specific formulation of combination OC used. The effect appeared to persist long after OC use had been discontinued; furthermore, protection increased with increasing cumulative duration of OC use.

The protective effect of OCs on endometrial cancer is most likely related to direct effects on the endometrium. Among current OC users the carcinogenic effect of unopposed estrogen on the endometrium is probably reduced because combination OCs contain both estrogen and progestin. The continued protection seen among past OC users is less well understood. Perhaps the combination of estrogen and progestin irreversibly changes endometrial cells so that they are not susceptible to carcinogens or to malignant transformation (CASH, 1987a).

Suppression of ovulation and suppression of pituitary secretion of gonadotropins have both been postulated as mechanisms by which OCs protect against ovarian cancer (Weiss, 1982). Two other factors that provide protection from ovarian cancer, increasing parity and breastfeeding (Gwinn et al., submitted), may also derive their protective effects from one of these two proposed mechanisms. Available epidemiologic studies do not provide sufficient information to choose one of these postulated mechanisms over the other.

Fourteen epidemiologic studies have found a decreased risk of benign breast disease (BBD) associated with OC use, including both case-control and cohort studies (Stadel, 1986). Evidence suggests that OCs decrease the risk of fibrocystic disease and fibroadenoma diagnosed by biopsy as well as the risk of breast lumps observed clinically but not biopsied. Results from a large cohort study conducted in the United Kingdom by the Oxford Family Planning Association have provided especially useful information about the relationship between OC use and BBD (Brinton et al., 1981). The decreased risk of BBD seen among women who use OCs occurs primarily among current or recent users who have used them for 2 years or longer. The relative risk among women who have used OCs for more than 2 years compared with nonusers is about 0.6 for fibrocystic disease and about 0.5 for unbiopsied breast lumps. The relative risk of fibroadenoma among women who have used OCs for less than 2 years is about 0.4, which is essentially the same as the relative risk of 0.3 among women who have used OCs for 2 or more years. The decreased risk of BBD does not persist among past OC users who have not used OCs for more than 1 year.

Many epidemiologic studies have found that a history of BBD increases a woman's risk of breast cancer. Even though OCs decrease the risk of BBD, epidemiologic studies have not found that OCs decrease the risk of breast cancer, as might be suggested by the OCs–BBD relationship. The most likely explanation for this paradox is that OCs probably decrease the risk of the large proportion of BBD that is not closely linked to breast cancer risk but do not decrease the risk of the types of BBD that increase a woman's risk of breast cancer (Stadel, 1986). Clearly, more information about the interrelationship between OC use, histologic types of BBD, and breast cancer is needed.

Seven epidemiologic studies have found that current or recent OC use reduces the risk of pelvic inflammatory disease (PID) (Stadel, 1986). On average, these studies have found that the risk of PID among OC users is about 40 percent lower than the risk among women using no contraceptive method.

The most detailed analysis of this issue comes from the U.S.-based Women's Health Study, a large hospital-based case-control study conducted from 1976 to 1978 (Rubin et al., 1982). This study found that the overall relative risk of PID among current OC users was about 0.5 compared with women using no contraception. However, this protective effect was limited to women who had been using OCs for at least 1 year. Furthermore, women not currently using OCs but who had used them in the past were no longer protected.

Two mechanisms have been proposed to explain how OCs protect against the development of PID. First, OCs may change cervical mucus so that it prevents pathogenic organisms from ascending into the upper genital tract. Second, because OCs reduce menstrual blood flow, a decreased amount of medium may be available for bacterial growth (Rubin et al., 1982).

Most of the studies of the relationship between OCs and PID have been hospital-based studies, so that case groups in these studies consisted of women hospitalized for PID. Because many women diagnosed with acute PID are not hospitalized, findings about women who are may not be generally extended to women who develop asymptomatic PID or symptomatic PID that does not require hospitalization (Washington et al., 1985). Specifically, gonorrhea may be an important cause of PID that requires hospitalization, whereas other bacterial etiologies such as chlamydia may cause much of the PID among women who do not require hospitalization. If OCs only protect against the bacterial etiologies of PID that are likely to require hospitalization, using OCs may not protect against some important causes of PID. Little epidemiologic evidence exists to clarify this question.

Results from a large cohort study in the United Kingdom have provided clear evidence that OC use decreases the risk of iron-deficiency anemia, in both current and past OC users (Royal College of General Practitioners, [RCGP], 1970). The protective effect provided by current OC use is probably due to the decrease in menstrual blood flow routinely seen among OC users. An increase in iron reserves probably accounts for the persistence of the decreased risk in past users. In countries where the prevalance of iron-deficiency anemia is high, this benefit to OC users may be especially important (Stadel, 1986).

Three epidemiologic studies have found that OC use decreases the risk of functional ovarian cysts, including follicular, granulosa lutein, and theca lutein cysts (Stadel, 1986). Decrease in risk appears to be confined to current OC users and is probably related to the suppression of ovulation that occurs during OC use.

Evidence from a case-control study that used data collected from the Oxford Family Planning Association cohort study suggests that OC use protects a woman from developing uterine fibroids (Ross et al., 1986). The risk of fibroids decreased with increasing duration of OC use: each 5 years of OC use contributed another 17 percent reduction in fibroid risk. The mechanism of the protective effect is still speculative. The authors proposed that circulating estrogens, either exogenous or endogenous, may promote the formation of fibroids and that the decreased risk associated with OC use may be explained by the modifying effect of the progestins in OCs.

Adverse Health Effects

Cardiovascular Effects

Most epidemiologic evidence suggests that OC use increases the risk of cardiovascular disease, in particular the risk of venous thromboembolism, myocardial infarction (MI), and stroke (Stadel, 1986). However, the risk of serious illness or death from cardiovascular disease that can be attributed to OC use is apparently concentrated among certain groups of women, primarily older women and women who smoke cigarettes.

At least 11 case-control and 4 cohort studies have found that OC use increases the risk of venous thromboembolism (Vessey, 1980). Results from those studies have shown that current OC use increases the risk of venous thromboembolism, although the increased risk does not appear to persist among past users. Furthermore, the risk among current users remains constant with increasing duration of OC use. The risk of both superficial and deep vein thrombosis among current OC users is directly related to the estrogen content of OCs: the higher the estrogen content of the OC, the greater the risk of venous thromboembolism (Stadel, 1986). The pathogenesis of venous thromboembolism among OC users probably involves an increase in the size of intravascular clots formed in response to thrombotic stimuli, most likely a result of estrogen-induced decreases in antithrombin III and plasminogen activators. Unlike the associations between OC use and MI and stroke, available studies have not found any interrelationship between OCs, venous thromboembolism, and cigarette smoking. The increased risk of venous thromboembolism is an important source of illness attributable to OC use but is a very infrequent cause of mortality (Stadel, 1986).

In contrast to the low attributable risk of death from venous thromboembolism associated with OC use, the increased risk of MI and stroke observed in women currently using OCs has been demonstrated to be an important source of the mortality risk attributable to OCs (Stadel, 1986). Current OC use increases the risk of MI, thrombotic stroke, and hemorrhagic stroke. The risk of MI and stroke associated with current OC use is strongly influenced by age and by the presence of other cardiovascular risk factors, such as cigarette smoking, hypertension, and diabetes. For example, the risk of MI that is attributable to OCs among nonsmoking women 30 to 39 years of age is about 4 cases per 100,000 current users per year, but it increases to about 185 cases per 100,000 current users per year among women aged 40 to 44 years who smoke heavily (Table 3). The risk attributable to past OC use appears also to be concentrated among older women and older women who smoke heavily. The risk of adverse cardiovascular events among current OC users appears to be directly related to the estrogen content of the OCs; although less conclusive, risk may also be related to progestin content. The pathogenesis of MI and stroke among current OC users may be related both to the

TABLE 3 Current Use of Oral Contraceptives (OCs), Cigarette Smoking, and Risk of Myocardial Infarction (MI)

Age	Cigarettes per Day	MIs per 100,000 Women per Year OC Users	MIs per 100,000 Women per Year Nonusers	MIs per 100,000 Current OC Users per Year Relative Risk[a]	MIs per 100,000 Current OC Users per Year Attributable Risk
30-39 yr	All women	11	4	3	7
	0-14	6	2	3	4
	>15	30	11	3	19
40-44 yr	All women	89	22	4	67
	0-14	47	12	4	35
	>15	246	61	4	185

[a] Relative risk of MI for OC users compared with nonusers.

Source: Modified from Stadel, 1986, p. 17.

intravascular coagulation system and to the effects of increased blood pressure and metabolic changes.

Current OC use has been found to elevate blood pressure slightly in most women—about 1 to 2 mm Hg diastolic and 5 mm Hg systolic (Stadel, 1986). OC use leads to approximately a threefold to sixfold increased risk of overt hypertension. This risk has been observed to increase with increasing age and with increasing duration of OC use. Whether other risk factors for hypertension may be related to the increased risk attributable to OC use has not be established.

Metabolic Effects

The progestin component of OCs has been found to decrease the concentration of high-density lipoprotein-cholesterol (HDL-C), whereas the estrogen component has been found to increase HDL-C concentration (Stadel, 1986). Hence, the effects of different OC formulations on HDL-C concentration apparently depend on the specific estrogen-progestin content. A U.S. study provided information about 10 combination OCs: 3 lowered HDL-C concentrations, 2 had no effect, and 5 increased HDL-C levels (Bradley et al., 1978). If the progestin component has a strong anti-estrogen effect, the tendency of the estrogen component to increase HDL-C concentration may be overpowered (Stadel, 1986).

Current OC use has been found to decrease glucose tolerance among most women, although this decrease appears to be small, unrelated to duration of use, and only additive to the effects of other risk factors for impaired glucose tolerance (Stadel, 1986). This decrease in glucose tolerance is directly related to the estrogen content of the OCs, although there may be a relationship to the progestin content also. More important, OCs containing relatively small amounts of estrogen—< 50 micrograms of ethinyl estradiol—do not appear to decrease glucose tolerance to any appreciable extent.

Neoplastic Diseases

Epidemiologic studies clearly indicate that OC use increases the risk of hepatocellular adenoma (HCA), a rare, benign neoplasm of the liver. Although benign, HCA can cause serious abdominal hemorrhage and death, with a death-to-case ratio of approximately 8 percent (Rooks et al., 1979) Among women of reproductive age who have never used OCs or who used them for a short time, HCA develops at an annual rate of about 1.0 to 1.3 per million women 16 to 44 years of age. However, OC use is a strong risk factor for HCA, with a relative risk greater than 100 among women who have used OCs for 3 or more years compared with women who have used OCs for 1 year or less. The increased risk appears to be directly related to the duration of use, the age of the user, and the estrogen content of the OC. However, the absolute risk of HCA associated with OC use is small because of the rarity of the tumor. Among women who have used OCs for 5 years or longer, the attributable risk is estimated to be about 2 cases of HCA per 100,000 users per year (Stadel, 1986).

Epidemiologic studies have well demonstrated that OC use protects women from developing endometrial and ovarian cancers. However, the effect OCs may have on the risk for developing certain other malignancies remains unclear. Hepatocellular carcinoma and malignant melanoma have both been associated with OC use, although the strength of the associations has not been great. For this reason as well as the fact that those tumors are quite rare, the public health impact of a true positive association would not be great for either type of malignancy. Conversely, the debate about whether OC use increases the risk of cervical and breast cancers remains heated; some studies have found no effect on cancer risk, while others have found disturbing increases in risk. Because breast and cervical cancers are two of the most common cancers affecting women, contraceptive providers and epidemiologists feel an urgent need to resolve these discrepant results. However, as will be discussed subsequently, the possibility of a quick resolution to the controversies is unlikely.

Both case-control and cohort studies have assessed the relationship between OC use and malignant melanoma. Generally, they have not found substantial increases in the risk of melanoma associated with OC use (Stadel, 1986). Certain studies do suggest that OCs may increase the risk among certain subgroups of women, especially those who have used OCs for a long time (Ramcharan et al., 1981; Bain et al., 1982; Holly et al., 1983; Beral et al., 1984). Few studies have adequately addressed the risk by histologic type of melanoma. Future studies of this association will have several issues to consider, including the rarity of the tumor among women, the different histologic subtypes of melanoma, and the potentially confounding effects of exposure to sunlight.

The association between OC use and benign liver tumors, as well as a number of case reports of liver cancer among OC users, have led to theoretical concerns that OC use might increase the risk of malignant liver tumors. Three case-control studies published since 1983 have found increased risks of hepatocellular carcinoma among OC users (Henderson et al., 1983; Forman et al., 1986; Neuberger et al., 1986). Generally, the increased risk has been confined to women with a history of long-term OC use. However, each of the studies had few women in the case group (≤30) and had methodological problems that may have biased the results.

In developed countries hepatocellular carcinoma is extremely rare among reproductive-aged women. In the United States in 1982 only 59 women died from liver cancer among approximately 52 million women aged 15 to 44 years (National Center for Health Statistics, 1982). Hence, even if OC use substantially increases the relative risk of liver cancer, the attributable risk would still be very low.

In many developing countries liver cancer is a much more common problem, primarily because of the relationship between hepatocellular carcinoma and chronic hepatitis B virus infection, which has a high prevalence in some regions. In those areas the possible interrelationships between OC use, hepatitis B infection, and liver cancer are more troublesome. Currently, the World Health Organization (WHO) is conducting a multicenter case-control study to address the relationship between OC use and liver cancer. Data are being collected from three developing countries with high rates of hepatitis B infection and liver cancer. It is hoped that results from this study will shed light on the relationship between OC use and this serious malignancy.

The potentially positive association between OC use and cervical cancer has added importance when considered in the setting of less developed countries. Surveillance information from developing countries, although sometimes fragmentary and incomplete, suggests that cancer of the cervix is the most frequent malignancy among women in those countries (Lunt, 1984). Unfortunately, screening efforts in those countries usually reach very limited segments of the female population. Unlike the situation in developed countries where provision of contraceptive services is usually accompanied by routine Papanicolaou (Pap) screening for cervical cancer, family planning programs in developing countries often do not have the resources to provide Pap screening for their clients.

To date, no definite causal relationship has been established between OC use and cervical cancer. Of 15 major epidemiologic studies, 8 have found no increased risk of cervical neoplasia and 7 have found significantly increased risks overall or increases among certain subgroups of users (Piper, 1985; Brinton et al., 1986; Ebeling et al., 1987; Irwin et al., 1988). At least some of the discrepancies may be due to methodological problems encountered in the various study designs, including (1) confounding bias caused by the effects of certain sexual behaviors that are well-recognized risk factors for cervical cancer; (2) overdiagnosis (by Pap screening) of cervical neoplasia among OC users; (3) a detection bias caused by increased Pap screening of OC users compared with nonusers; and (4) inappropriate comparison groups that include women who have used barrier methods, which are thought to protect against cervical cancer. (Swan and Pettiti, 1982; Piper, 1985) One recent case-control study from Costa Rica that considered the effects of sexual activity, history of sexually transmitted diseases (STDs), and the enhanced detection of cervical neoplasia among OC users found no elevated risk of invasive cervical cancer associated with OC use (Irwin et al., 1988). Although women who had used OCs had an increased risk of carcinoma in situ compared with never-users in the study, this increased risk was confined to women who had recently used OCs. Further, no increased risk of carcinoma in situ was found among subgroups in whom a history of Pap screening was not strongly linked to OC use. Both findings suggest that any elevated risk of carcinoma in situ among OC users may have been due to a bias caused by enhanced detection of disease. Another study of invasive cervical cancer from Maryland found no association with OC use (Celentano et al., 1987). However, two other recent studies, which also controlled for potentially confounding factors, found a 50 percent increased risk of invasive cervical cancer among women who had ever used OCs; women who used OCs for 5 or more years had about a twofold increase in risk (Brinton et al., 1986; Ebeling et al., 1987).

Results from these recent studies of the OC–cervical cancer relationship remain conflicting, even though most of the potential methodological problems were considered. OCs probably do not dramatically increase the overall risk of cervical dysplasia or cancer, although long-term use or use by specific subgroups of women may increase the risk. Furthermore, little is known about the relationship between OC use and human papillomavirus infection, which is thought to have an important role in cervical carcinogenesis. Further research is needed to clarify these complex relationships. Certainly, OC users, both current and past, need to be screened regularly with Pap smears. Whether or not using OCs is a risk factor for cervical cancer, it provides an ideal opportunity to screen sexually active women for this serious reproductive system cancer.

More than 15 published studies have reported that, overall, use of OCs does not appear to increase or decrease a woman's risk of breast cancer (Prentice and Thomas, 1987). However, many of those investigations were conducted in the 1970s, little more than 10 to 15 years after OCs were introduced. In these earlier investigations researchers had limited ability to study the long-term effects of OC use, particularly the effects of long duration of use, of distant time since first use, of use at specific ages, and of use in relation to other key reproductive events related to a woman's risk of developing breast cancer. Additionally, many of the studies of the OC–breast cancer relationship were not large enough to examine the relationship according to specific OC formulations and among certain high-risk subgroups of women.

To date, the CASH Study is the largest study designed to assess the risk of breast cancer associated with OC use (CASH, 1986). The study was a population-based case-control study conducted in eight regions of the United States from 1980 to 1982. Detailed information about reproductive history, contraceptive use, family history of cancer, and personal characteristics and habits was collected from 4,711 women with newly diagnosed breast cancer and from 4,676 control women. Case and control women were from 20 to 54 years of age. Compared with women who had never used OCs, women who had used OCs had a relative risk of breast cancer of 1.0 (0.9–1.1); even women who had used OCs for 15 or more years had no increased risk. None of the 12 OC formulations most commonly used in the United States were associated with a statistically significant increased risk of breast cancer. Analyses also showed no increase in risk among high-risk subgroups of women, including women with a history of BBD or a family history of breast cancer or those who were nulliparous or older at first term pregnancy.

Nonetheless, despite many studies indicating that OC use apparently does not increase the overall risk of breast cancer, controversy remains concerning whether long-term OC use, use at an early age, or use before the first term pregnancy might increase breast cancer risk (Skegg, 1988). Several recent studies have found increases in risk among certain subgroups of women. An analysis published in 1983 suggested that women who used certain OC formulations classified as "high progestin" before the age of 25 appeared more likely to develop premenopausal breast cancer than women who did not use OCs before age 25 (Pike et al., 1983). In the same year a report from England suggested that women with long-term OC use before their first child was born had an increased risk of premenopausal breast cancer compared with women who did not use OCs before the birth of their first child (McPherson et al., 1983). A 1985 report described results from an analysis using a subset of data from the CASH Study that replicated the analyses from the two 1983 reports (Stadel et al., 1985). In this analysis 2,088 women who had breast cancer and were from 20 to 44 years old were compared with 2,065 control women in the same age group. No significant increase or decrease in risk was observed among women who used high-progestin OCs before age 25, even when duration of use exceeded 6 years. Similarly, women who used OCs before their first term pregnancy did not have an increased risk, even when duration of use exceeded 4 years.

In 1986 two additional conflicting reports were published (Meirek et al., 1986; Paul et al., 1986). Analyses from a case-control study conducted in Scandinavia found a risk of premenopausal breast cancer among women with long-term OC use and use before the first term pregnancy (Meirek et al., 1986). The investigators reported a twofold increase in risk of breast cancer among women who used

OCs for as long as 12 years. In contrast, analyses from a New Zealand case-control study did not find elevations in risk associated with OC use, even among women who had used OCs for a long time and who started taking them at an early age or among women who used OCs before their first term pregnancy (Paul et al., 1986).

A recently published analysis from the CASH Study examined in detail the risk of breast cancer in relation to early use of OCs (Schlesselman et al., 1988). The study found no evidence that use of OCs in the distant past increased breast cancer risk through age 54. Parous women who had used OCs for more than 6 years before their first term pregnancy, at 10–14 years after that pregnancy, were found to have a risk of breast cancer relative to nonusers of 1.1 (0.3–3.9). Among nulliparous women with more than 6 years of OC use, the relative risk of breast cancer at 10–14 years after they last used OCs was 0.6 (0.1–3.7).

Epidemiologists continue trying to disentangle the reasons for the discrepancies among the published studies (McPherson et al., 1986; McPherson and Drife, 1986; Anonymous, 1986; Schlesselman et al., 1987; Skegg, 1988). McPherson et al. (1986) have suggested that any possible risk of breast cancer associated with OC use at early ages may not become apparent for at least 20 years. Women born in the 1940s who used OCs in the 1960s will reach the ages of highest risk for breast cancer by the year 2000. If OC use at young ages is a risk factor for breast cancer, and if at least 20 years is required between exposure to OCs and the diagnosis of breast cancer, then these women might be expected to be diagnosed with breast cancer some time during the next 20 years. Under these assumptions, researchers may not be able to detect such a relationship at the present time.

The conflicting reports about the possible relationship between OC use and breast cancer risk, particularly use at an early age and before first term pregnancy, are confusing and troublesome. However, the preponderance of epidemiologic studies suggests that, overall, OCs do not increase the risk of breast cancer. If they do increase the risk in certain subgroups of women, the increase is not great and is generally confined to long-term users. Most experts believe that current recommendations for OC use should not be changed because of the continuing OC-breast cancer controversy. As women who used OCs in the 1960s reach the ages of highest risk for breast cancer, investigators should vigorously pursue opportunities to continue studying the long-term effects of OC use on breast cancer risk.

Other Effects

Several epidemiologic studies published in the 1970s reported that using OCs increased a woman's risk of gallbadder disease. Results from two large cohort studies from the United Kingdom published in 1982 better clarified the relationship between OCs and gallbladder disease (Layde et al., 1982; RCGP, 1982). Each study found that the increased risk appeared to be confined to recent users and to short-term users. No differences in risk were found with differing estrogen or progestin doses. The authors of both studies concluded that using OCs apparently accelerated the development of gallbladder disease among susceptible women, rather than increased the overall lifetime risk.

A twofold to threefold increase in the prevalence of chlamydia trachomatis infection of the cervix among OC users has been demonstrated in 12 epidemiologic studies (Washington et al., 1985). Three reasons for this increase in risk have been suggested: (1) greater sexual activity among OC users than nonusers, (2) enhanced detection of chlamydial infection because of increased cervical ectropion induced by OC use, and (3) a direct effect of OC use on the risk of chlamydial infection. Three of the epidemiologic studies measured sexual activity and found no increase among OC users; furthermore, several epidemiologic studies have not found an increased risk of gonococcal infection among OC users (Washington et al., 1985). Whether or not it is merely easier to detect chlamydia infection among women who use OCs cannot be easily assessed and remains unresolved.

INTRAUTERINE DEVICES

The first IUDs that were widely used, such as the Lippes Loop™ and Saf-T-Coil™, were made of inert plastic. In the 1970s a second generation of IUDs was introduced in which the plastic IUD acted as a carrier for metal or hormonal substances (Piotrow et al., 1979). These medicated devices included the Copper-7, Copper-T series, and the Progestasert™. More recently a new and improved copper IUD, the Copper-T 380A, was developed; it may soon be the major IUD available in most countries (Treiman and Liskin, 1988).

The IUD is a highly effective contraceptive, with method failure rates of about 1 percent per year for medicated IUDs and 2 percent per year for nonmedicated ones (Table 2). Because of undetected IUD expulsion, the user failure rate for IUDs is estimated to be somewhat higher, about 6 percent per year.

Because IUDs apparently prevent both intrauterine and ectopic pregnancies, the overall risk of ectopic pregnancy among IUD users is probably decreased compared with women who use no contraception. A 1981 study reported that current IUD users had a relative risk of ectopic pregnancy of 0.4 (0.3–0.6) compared with women using no contraception (Ory, 1981). However, about 5 percent to 15 percent of IUD-associated pregnancies are ectopic as IUDs seem to be more effective at preventing intrauterine pregnancies.

No major noncontraceptive health benefits are linked to IUD use. Progesterone-elaborating IUDs tend to decrease menstrual blood loss and dysmenorrhea, which can be viewed as a benefit for those women in whom it occurs (Hatcher et al., 1988).

Adverse Health Effects

Four major health risks have been associated with IUD use: (1) spontaneous abortion, which may rarely progress to septic abortion; (2) uterine perforation; (3) PID; and (4) tubal infertility. Based on information from studies done primarily in the United States and other developed countries, estimates for the annual mortality risk attributable to IUD use are between 1 and 2 deaths per 100,000 women (Ory et al., 1983). Mortality rates from IUD use may be somewhat higher in developing countries because of delays in treating complications or lack of access to medical facilities.

Although pregnancy rates are low among IUD users, complications may occur in women who do become pregnant. If the IUD is left in place, the chance of spontaneous abortion is 50 percent. If the IUD is removed, this rate drops to 25 percent (Hatcher et al., 1988). Septic abortion is a rare but sometimes fatal complication (10 per 100,000) that may occur if the IUD is left in place after the first trimester of pregnancy (Ory et al., 1983). If the IUD is removed as soon as the pregnancy is diagnosed, most instances of septic abortion can be prevented.

Perforation of the uterus may occur when an IUD is inserted, although this injury is often undetected and usually not serious. The incidence of perforation is unknown but is probably less than 1 percent (Hatcher et al., 1988). Very rarely the IUD is extruded into the abdominal cavity, requiring laparotomy to remove it. The risk of perforation during insertion increases substantially in two subgroups of women: lactating women and women within 8 weeks of delivery, because they have softer uterine musculature; at these times the risk of perforation may be increased substantially (Heartwell and Schlesselman, 1983).

Most epidemiologic studies of the issue have found that IUD users have an increased risk of PID (Grimes, 1987). This finding contrasts with all other modern methods of temporary contraception, which seem to protect a user from developing PID.

PID is usually but not always the result of an STD, such as gonorrhea or chlamydial infection. The overall incidence of PID varies greatly in different populations and depends mainly on sexual behaviors. The incidence of PID among IUD users also varies substantially. In a Scandinavian study of women with gonorrhea, 24 percent of IUD users developed PID (Ryden et al., 1979). Results from a large cohort study of married women in the United Kingdom showed a PID rate of 0.15 per 100 woman-years (Vessey et al., 1981). Clinical trials that determined rates of IUD removal because of PID found rates about eight times greater than the study from the United Kingdom, up to 1.22 per 100 woman-years (Siven and Stern, 1979).

The Dalkon Shield™, an IUD used in the 1970s but no longer available, has been associated with a high risk of PID in several studies (Grimes, 1987). Women using IUD types other than the Dalkon Shield have been found to have about 1.5

to 2.0 times greater risk of PID than women using no contraceptive method. Most studies that have looked at the relationship of PID risk to the timing of IUD insertion have found that much of the increased risk is probably confined to the first few months after insertion. A recently published analysis has found that IUD users who are in mutually monogamous sexual relationships probably have little increased risk of PID associated with their IUD use (Lee et al., 1988). Much of the increased risk of PID seen among IUD users may be confined to women who are at increased risk for developing STDs.

One of the most important and serious sequellae of PID is tubal infertility. Results from a large cohort study of women with surgically confirmed PID have demonstrated that these women have an increased risk of subsequent tubal infertility (Westrom, 1987). The data showed that, among the women with PID who were subsequently exposed to a chance of pregnancy, infertility occurred in 11 percent after one episode of PID, 23 percent after two episodes, and 54 percent after three episodes.

Until recently little epidemiologic evidence directly linked IUD use with infertility. Results from most clinical trials of IUDs found that more than 70 percent of women conceive within 12 months of IUD removal (Liskin and Fox, 1982).

A large cohort study from the United Kingdom found that within 2 years of removal 92 percent of former users had given birth (Vessey et al., 1978). However, two U.S. case-control studies reported that the risk of tubal infertility among nulliparous women who had ever used IUDs was about twice that of those who had never used them (Daling et al., 1985; Cramer et al., 1985).

Both of these case-control studies found different risks of tubal infertility associated with different types of IUDs (Daling et al., 1985; Cramer et al., 1985). Women who had used the Dalkon Shield had the highest risk of tubal infertility. The lowest risk was seen among women who had used copper IUDs, while women who used the Lippes Loop or Saf-T-Coil IUDs had intermediate risks. Although these findings were consistent between the two studies, the numbers of users of specific IUD types were small, and the differences may have been due to chance alone.

The incidence of IUD-associated infertility is unknown, although it is probably less than the incidence of IUD-associated PID. The increased risk of infertility is presumably related to the increased PID risk associated with IUD use, even if PID is never recognized clinically. Cramer et al. (1985) found that women who reported having only one sexual partner had no increased risk of tubal infertility associated with IUD use.

BARRIER METHODS

Because of the potential for preventing transmission of STDs, such as acquired immune deficiency syndrome, researchers are focusing new attention on barrier methods of contraception—condoms, diaphragms, spermicides, and sponges. Generally, barrier methods are less effective at preventing pregnancy than are OCs, IUDs, sterilization, injectables, and implants. Further, the effectiveness of barrier methods is extremely user dependent; improper use by unmotivated users can result in high failure rates. Side effects are minimal; consequently, the main risks associated with barrier methods are complications from unintentional pregnancy (Ory et al., 1983).

Discontinuation of use of barrier methods is common, both because they have higher risks of failure and because they may be obtrusive and less convenient to use (Sherris et al., 1984). But an important advantage of barrier methods is their protection against various STDs. Barrier methods also protect against acute PID and the resulting tubal infertility by keeping organisms that cause PID—such as Neisseria gonorrhoeae and Chlamydia trachomatis—from infecting the lower genital tract and from ascending into the upper genital tract (Kelaghan et al., 1982; Cramer et al., 1987).

Condoms

Condoms are a safe, reversible method of birth control increasingly being used to prevent both pregnancy and STDs. The estimated method failure rate for condom use (without spermicide) is 2 percent per year, while the user failure rate is 12 percent (Table 2). The effectiveness of condoms as both a contraceptive and a disease prophylactic depends on consistent and proper use. If couples begin to rely solely on condoms for protection against both pregnancy and infection, they could experience a substantial increase in the number of accidental pregnancies from incorrect or inconsistent use (Trussell and Kost, 1987). As condom use increases, acceptability and compliance will be important to monitor.

In vitro studies have demonstrated that latex condoms are effective barriers to herpes simplex virus type 2 (Conant et al., 1984; Judson et al., in press), Chlamydia trachomatis (Judson et al., in press), cytomegalovirus (Katznelson et al., 1984), and human immunodeficiency virus (HIV) (Conant et al., 1986; Rietmeijer et al., 1988). Natural membrane condoms may give less protection against viral STDs than latex ones, which contain smaller pores (Centers for Disease Control, 1988). Because using condoms prevents semen deposition, their use should also reduce transmission of organisms that may be present in semen, such as Neisseria gonorrhoeae, hepatitis B virus, Trichomonas vaginalis, and HIV (Stone et al., 1986).

Epidemiologic data relating to condom use and the prevention of STDs are limited. Several studies have shown that condom users, their partners, or both have a lower frequency of gonorrhea (Barlow, 1977; Hooper et al., 1978). Preliminary epidemiologic data suggest that using condoms may protect against sexual transmission of HIV infection (Fischl et al., 1987; Centers for Disease Control, 1987). Recent studies of prostitutes have also suggested a protective relationship between condom use and HIV infection (Smith and Smith, 1986; Centers for Disease Control, 1987; Mann et al., 1987).

However, these studies were based on observational data, and condom users are likely to differ from nonusers in important characteristics that may be related to the risk of acquisition of STDs (Feldblum and Fortney, 1988). Although conclusive epidemiologic evidence is lacking, available data suggest that the use of condoms is an effective preventive measure against STDs (Horsburgh et al., 1987). Failure of condoms to protect against STDs is probably explained by user failure more often than by product failure (Centers for Disease Control, 1988). The use of spermicides is considered a useful adjunct to condoms because spermicides can inhibit in vitro the growth of a broad range of sexually transmitted pathogens (Cramer et al., 1987; Francis and Chin, 1987).

Spermicides and the Contraceptive Sponge

Spermicides are chemical agents that inactivate sperm in the vagina before the sperm can move into the upper genital tract. Spermicides in use today include nonoxynol-9, octoxynol-9, and menfegol, although the latter is not available in the United States. Spermicides containing mercuric compounds, rejected as unsafe by the U.S. Food and Drug Administration (FDA), are still marketed in some Latin American countries and elsewhere (Sherris et al., 1984). Inert carriers of the spermicide include foams, jellies, creams, and suppositories; newer carriers include foaming suppositories (Neo Sampoon™) and spermicide-impregnated sponges (Today™).

The type of spermicidal agent or carrier has little effect on failure rates. The method failure rate is estimated to be about 3 percent per year; however, the user failure rate, at 21 percent per year, is much higher (Table 2), largely because use must be premeditated. Furthermore, because spermicides are messy and can be irritating to mucous membranes, women often stop using them (Sherris et al., 1984). Protection from pregnancy, however, can be increased considerably if spermicides are used in conjunction with other barrier methods such as diaphragms or condoms.

Among nulliparous women the failure rates for the contraceptive sponge are comparable to the failure rates for the diaphragm; the method failure rate is 5 percent per year and the user failure rate is 18 percent per year (Table 2). However, among parous women the method failure rate is 8 percent and the user failure rate is 28 percent.

Both laboratory evidence and clinical evidence suggest that spermicides protect against various STDs (Stone et al., 1986). In vitro studies have shown that spermicides inhibit the growth of Neisseria gonorrhoeae (Singh et al., 1972; Cowan and Cree, 1973) and inactivate herpes simplex virus type 2 (Singh et al., 1976) and HIV (Hicks et al., 1985). Although epidemiologic evidence is sparse, the virucidal effects of spermicides may provide some protection against cervical cancer, since the cancer may be initiated or promoted by a sexually transmitted virus such as human papilloma virus (Spring and Gruber, 1985). Several epidemiologic studies have found that spermicides protect users from gonorrhea infections (Jick et al., 1982; Quinn and O'Reilly, 1985), especially when used with condoms or diaphragms (Austin et al., 1984). A clinical trial among high-risk women using contraceptive sponges impregnated with nonoxynol-9 demonstrated protection against chlamydial and gonorrheal infections (Rosenberg et al., 1987).

One epidemiologic study suggested a connection between spermicides used near the time of conception or during pregnancy and the risk of congenital defects (Jick et al., 1981). Since that report, several larger and better designed studies have failed to confirm the association (Huggins et al., 1982; Mills et al., 1982; Shapiro et al., 1982; Cordero and Layde, 1983).

Additional health risks have been associated with the contraceptive sponge, a newer type of spermicidal carrier. Sponge users may be at increased risk of vaginal candidiasis, possibly because nonoxynol-9 allows candidal overgrowth (Rosenberg et al., 1987). Of greater health consequence is a possible association between using the contraceptive sponge and developing toxic shock syndrome (TSS); sponge users have a relative risk of 10.5 (2.1–52.7) compared with women using no barrier method (Schwartz et al., 1989). Because TSS is an extremely rare disease, the absolute risk of sponge users developing TSS is quite small; however, because TSS is a serious illness, physicians and sponge users should be aware of the symptoms and alert to the possibility (Faich et al., 1986; Schwartz et al., 1989). Postpartum women and women who have had TSS should not use the sponge, and women should never leave the sponge in the vagina for more than 30 hours (Reingold, 1986).

Diaphragm With Spermicide

Diaphragms used with spermicidal cream or jelly are a safe method of contraception and, when used correctly and consistently, can be an effective contraceptive. Because diaphragms/spermicides are inconvenient to use, only highly motivated women will experience low failure rates. Although the method failure rate is estimated to be about 3 percent per year, the user failure rate is much higher, about 18 percent per year (Table 2). Clinicians should consider how motivated their patients are before advising diaphragm use (Sherris et al., 1984).

As with other barrier methods, diaphragm/spermicide use may have several noncontraceptive health benefits. This method appears to protect against gonorrhea, PID, and tubal infertility (Jick et al., 1982; Kelaghan et al., 1982; Cramer et al., 1987). Several case-control studies found cervical dysplasia and cervical neoplasia less common among diaphragm users than among other women (Wright et al., 1978; Harris et al., 1980; Celentano et al., 1987). Because diaphragms are almost always used with spermicides and spermicides alone may protect against various STDs, the separate protective effects of diaphragms on specific conditions may be difficult to measure (Stone et al., 1986).

As with the contraceptive sponge, diaphragms have been associated with a significantly higher risk of TSS (Hymowitz, 1981; Baehler et al., 1982; Schwartz et al., 1989). In one case-control study diaphragm users had a relative risk of TSS of 11.7 (2.5–56.1) compared with nonusers; however, the estimated absolute risk of TSS associated with diaphragm use was low, about 2.25 cases per 100,000 users per year (Schwartz et al., 1989). A less serious but more frequent complication associated with diaphragm use is urinary tract infections; the relative risks for diaphragm users developing a urinary tract infection compared with nonusers ranges from 2.0 to 3.0 (Foxman and Frerichs, 1985; Fihn et al., 1985; Vessey et al., 1987). Better fitted diaphragms and urination after intercourse may reduce the risk of urinary tract infections (Foxman and Frerichs, 1985). Women at particular risk for urinary tract infections, such as those with recurrent infections, should probably consider other contraceptive methods.

Cervical Caps

Cervical caps are cup-shaped devices, held in place by suction, that fit over the cervix (Hatcher et al., 1988). When used with spermicides, their effectiveness is comparable to that of diaphragms, with a method failure rate of about 5 percent per year and a user failure rate of 18 percent per year (Table 2). Cervical caps have not been widely used; hence, little published research is available. Reports from small studies suggest that about half the women who want to use the cap cannot be fitted. Many other women discontinue use because of odor problems, difficulty in insertion or removal, or dislodgement during intercourse (Ory et al., 1983). Advantages are that the cap can be left in place longer than 24 hours and that it can be inserted many hours or even a day or two before coitus (Sherris et al., 1984). Although potential health risks and benefits associated with the cervical cap are probably similiar to those associated with the diaphragm, available data are too limited to document them (Hatcher et al., 1988).

LONG-ACTING METHODS

Several long-acting contraceptive methods now available are highly effective, convenient to use, and give protection from pregnancy for 1 month to 5 years. All contain some kind of progestin. Disturbance of the menstrual cycle is the most common side effect of all progestin-containing contraceptives and is the major reason women stop using them.

Injectables

Two long-acting injectable progestins—depot-medroxprogesterone acetate (DMPA; Depo-Provera™) and norethindrone enanthate (NET; Noristerat™)—

are the most widely used and studied of several injectable contraceptives available. DMPA is approved for contraception in at least 90 countries, NET in more than 40 (Liskin et al., 1987). At present neither is licensed for use as a contraceptive in the United States.

Injectables are highly effective: the estimated method failure rate is 0.3 percent per year for DMPA, given every 90 days, and 0.4 percent per year for NET, given at 8- or 12-week intervals (Table 2). Because minimum compliance is necessary, the user failure rates are approximately equal to the method failure rates. Injectables prevent pregnancy in several ways. Possible mechanisms include inhibiting ovulation; thickening cervical mucus; and altering the endometrial lining, which inhibits implantation (Liskin and Quillin, 1982).

Considerable controversy regarding possible cancer risks has been associated with DMPA and NET, chiefly stemming from data from animal studies suggesting that DMPA use could increase the risk of breast and endometrial cancers (WHO, 1986a). Although the applicability of these animal models is debatable and human studies have either failed to demonstrate these relationships or are inconclusive (Liang et al., 1983; WHO, 1986a; Lee et al., 1987), the possibility of increased risk of cancer among DMPA users was one reason the FDA did not approve DMPA for contraceptive use in the United States (Sun, 1984).

In 1986 preliminary results were published from an ongoing case-control study conducted by WHO concerning the relationship between DMPA and breast cancer (WHO, 1986a). This is the largest epidemiologic study of the DMPA–breast cancer relationship published to date. Women who had ever used DMPA had a risk of breast cancer of 1.0 (0.7–1.5); no increase in risk was seen even for long-term users. Conversely, a recent study in Costa Rica found that DMPA users had an elevated risk of breast cancer of 2.6 (1.4–4.7); however, no dose-response effect was found (Lee et al., 1987). Because of the small number of DMPA users in the study and the lack of a dose-response relationship, the authors considered the results to be inclusive.

Another analysis from the same WHO study found that the risk of invasive cervical cancer was slightly higher for long-term DMPA users than for never-users (relative risk of 1.4 [0.9–2.2]); however, results were considered inconclusive because of the small number of long-term DMPA users as well as the lack of a consistent trend with duration of use (WHO, 1986a). Results from the Costa Rica study showed no increase in risk of either invasive cervical cancer or carcinoma in situ associated with DMPA use (Oberle et al., 1988).

Although DMPA and NET have yet to be conclusively linked to any human cancer, epidemiologic studies have been hindered by small sample sizes and short durations of exposure. Whether injectable contraceptives are associated with any cancer will be resolved only after additional data have accumulated (WHO, 1986a).

Reported metabolic effects of DMPA and NET use include changes in blood pressure as well as changes in insulin, cholesterol, and triglyceride levels; however, the findings are inconsistent and have shown no clear clinical significance

(WHO, 1986b; Liskin et al., 1987). Concern over cholesterol levels among women who use DMPA or NET is mainly due to the reported cardiovascular problems associated with OC use, generally thought to be caused by the estrogen component of the pill (Rosenfield et al., 1983). Lower levels of HDL-C have been reported among women who were taking OCs with a high progestin potency or a progestin alone (Bradley et al., 1978). Most studies of DMPA users have found either no change in total cholesterol and triglycerides or a decrease, a possibly beneficial effect (Liskin et al., 1987). However, a few studies have reported an increase in cholesterol with longer use of DMPA or a decrease in HDL-C, both possibly adverse effects. The only study involving NET also found a decrease in HDL-C (Fotherby et al., 1982). The clinical significance of these decreases in HDL-C is uncertain; to date, no associations between DMPA or NET use and increased incidence of cardiovascular disease have been reported. Trials are under way in five countries to measure changes in lipid metabolism in DMPA and NET users (Liskin et al., 1987).

Unlike combination OCs, DMPA and NET appear to have little effect on the coagulation system (Liskin et al., 1987). Most laboratory studies have found little change in the coagulation and fibrinolytic systems that affect blood clotting.

The most consistently reported side effect of DMPA and NET use is a change in menstrual pattern: more than two-thirds of DMPA users and one-half of NET users have no regular menstrual cycles during the first year they use these contraceptives (Liskin et al., 1987). Amenorrhea or irregular, unpredictable bleeding episodes are the most common problems (WHO, 1978, 1987) and the principal reasons that women stop using injectables. Amenorrhea, more frequent with DMPA than NET, is probably the most upsetting change because women cannot be sure they are not pregnant (Liskin et al., 1987). Prolonged bleeding or spotting can be disturbing, especially if local customs restrict the activities of menstruating women. The occurrence of heavy bleeding requiring medical attention is rare, found among about 0.5 percent of users. In fact, because bleeding is often lighter than normal, increased hemoglobin levels among some users have been reported (WHO, 1986b).

Studies of children exposed to DMPA from breast milk have found no measurable consequences, even when followed up to 10 years after exposure (Liskin et al., 1987). This is not an unexpected finding, for very little of the hormone is transmitted in breast milk.

Neither DMPA nor NET appears to have any permanent effect on fertility (Liskin et al., 1987). However, resumption of ovulation and fertility is delayed; ovulation can be inhibited for 4 to 9 months or more after the last injection (Pardthaisong et al., 1980; Affandi et al., 1987). Although return to fertility is comparable to that seen with OC and IUD use, because of fears about permanent infertility, some family planning programs use injectables only among higher-parity women (Liskin et al., 1987). In fact, injectable progestins may protect women from PID, a major cause of infertility, by causing changes in cervical mucus (Gray, 1985).

Injectable progestins may also protect against endometrial and ovarian cancers. Excess estrogen is known to increase the risk of endometrial cancer, while progestins mitigate this effect (Rosenfield et al., 1983). Further, several studies have demonstrated a negative association between the use of combination OCs and endometrial cancer, a protective effect thought to be caused by the progestin component. Preliminary results from a WHO case-control study found that DMPA users had a relative risk of 0.3 (0.04–2.4) compared with never-users; however, data were based on just 52 women with endometrial cancer, only one of whom had ever used DMPA (WHO, 1986a).

Even fewer data exist on the association between ovarian cancer and DMPA use. However, several studies have demonstrated a negative association between OC use and ovarian cancer, perhaps due to the effects of anovulation (Rosenfield et al., 1983). Because injectable progestins usually prevent ovulation, there is at least a theoretical possibility that use of DMPA or NET might reduce the risk of ovarian cancer. Preliminary results from the WHO study support this possibility: DMPA users had a relative risk of ovarian cancer of 0.7 (0.3–1.7) compared with never users, based on 7 exposed cases and 74 exposed controls (WHO, 1986a).

Several new types of injectables are currently in use or under development. Injectable microspheres and microcapsules release hormone at a more constant rate, and preliminary trials have found fewer side effects than with DMPA or NET (Liskin et al., 1987). However, as with all progestational agents, irregular menstrual bleeding is a common side effect. Estrogen-progestin combinations that are injected monthly are being used in Latin America and China; preliminary results have shown highly effective protection and fewer problems with bleeding irregularities.

Implants

The NORPLANT® subdermal implant system is another highly effective, long-acting progestational contraceptive method (Liskin et al., 1987). It is currently being considered by the FDA for use in the United States. Developed by the Population Council, NORPLANT® implants come in two forms. The first and more widely used system, NORPLANT®, consists of six silastic rods, each about 1 inch long and 0.1 inch in diameter, containing 35 milligrams of the progestin levonorgestrel. The newer system, NORPLANT-2®, consists of two rods, each containing 70 milligrams of levonorgestrel. With both systems, rods are implanted under the skin, usually in the upper arm; the hormone is then released at a continuous, slow rate. Insertion and removal require a minor surgical procedure performed by trained health care personnel; however, once implanted, no further action is required for several years.

Both systems provide long-term protection from pregnancy. NORPLANT® is highly effective for 5 years, NORPLANT-2® for at least 3 years. In the first year of use the average reported failure rate is 0.2 percent (Table 2). However, the failure rate may be slightly higher among obese women. The mechanisms for pregnancy prevention are probably similar to those for the injectable contraceptives.

The rate of ectopic pregnancy among NORPLANT® users, about 1.5 per 1,000 woman-years, is about the same as the rate among women using copper and unmedicated IUDs (Liskin et al., 1987). Because the rate is lower than that for women using no contraception, NORPLANT® users can be considered to be protected from ectopic pregnancy.

As with injectables, the most common side effect of implants is disturbance of the menstrual cycle. Bleeding irregularity, the most frequent reason that women discontinue use of the method (Salah et al., 1987; Pasquale et al., 1987), causes 2 percent to 7 percent of women to stop using implants in the first year (Liskin et al., 1987). However, episodes of abnormal bleeding diminish with duration of use (Salah et al., 1987; Diaz et al., 1987), and the implants can be removed immediately if there are any disturbing side effects.

Transient ovarian cysts occur in a small percentage of women using NORPLANT® (Salah et al., 1987; Diaz et al., 1987); although the cysts regress eventually, they have been a reason for implant removal (Pasquale et al., 1987). Permanent infertility does not appear to be a concern; several studies have shown that fertility quickly returns after implants are removed (Diaz et al., 1987; Affandi et al., 1987).

Generally, no changes in several metabolic indicators have been found secondary to NORPLANT® use, including liver function, carbohydrate metabolism, blood coagulation, blood pressure, or body weight (Liskin et al., 1987). Studies of lipid metabolism in NORPLANT® users have produced conflicting results. However, no epidemiologic studies of cardiovascular complications among NORPLANT® users have been completed.

TUBAL STERILIZATION

Since the 1930s more than 95 million women worldwide have undergone tubal sterilization, making it the most widely used contraceptive method in the world (Liskin et al., 1985). The popularity of tubal sterilization continues to grow rapidly in many countries, which suggests that the estimate of the number of women who have had the procedure may be a conservative one. Currently, more than 15 percent of married women of reproductive age have undergone tubal sterilization in countries as diverse as Brazil, Panama, South Korea, Thailand, and the United States. In China an estimated 27 percent of women have undergone tubal sterilization. Tubal sterilization has been very popular in the United States, Latin America, the Caribbean, and in much of Asia. Africa and the Middle East have the lowest prevalence of tubal sterilization.

Tubal sterilization procedures can be characterized by (1) timing with respect to pregnancy, (2) the surgical approach to the fallopian tubes, and (3) the method of tubal occlusion. Tubal sterilization can be performed shortly after vaginal delivery, concurrently with cesarean section, at the time of induced abortion, or remote from pregnancy termination. In the latter instance the procedure is known as interval tubal sterilization.

Surgery for most tubal sterilizations requires an abdominal incision. For either postpartum or interval sterilizations, an abdominal incision 2 to 5 centimeters long, termed a minilaparotomy, allows the surgeon access to the fallopian tubes (Ross et al., 1985).

Laparoscopic tubal sterilization is becoming increasingly popular, particularly in developed countries. For laparoscopic procedures the abdominal incision is usually only about 1 centimeter long. Two general methods of inserting the laparoscope are used. In the first, termed *closed laparoscopy*, a surgeon uses an insufflating needle, a sharp trocar, or both to enter the abdominal cavity before inserting the laparoscope. An alternative technique called *open laparoscopy* attempts to minimize the risk of intra-abdominal injury caused by the insufflating needle or trocar by combining features of minilaparotomy and closed laparoscopy. Although open laparoscopy requires more time than closed laparoscopy, it reduces the chance of trauma to major blood vessels and the gastrointestinal tract and thus is inherently safer, especially for facilities that do not have the equipment and personnel necessary to handle serious surgical complications.

Alternative surgical approaches include the vaginal approach and the transcervical approach. Although the vaginal approach produces no visible scar, it is used less frequently because of the increased risk of pelvic infection (Ross et al., 1985). A transcervical approach, involving instillation of a sclerosing agent into the fallopian tubes, is widely used only in China.

The fallopian tubes can be occluded by ligation (with or without resection), by coagulation using unipolar or bipolar current, or by mechanical occlusion with silastic bands or clips. Tubal occlusions for postpartum laparotomies are generally done by ligation. All of these techniques can be used for interval sterilization, but the methods of occlusion for laparoscopy are limited to the use of coagulation or mechanical devices. Unipolar coagulation involves use of an electrical current that passes into the tube and through the patient to a ground plate attached to the patient. This system, which has resulted in fatal thermal bowel injuries (Peterson et al., 1981a), has largely been replaced in the United States by a bipolar coagulation technique that includes only the tissue in the grasping forceps as an integral part of the electrical circuit, thereby reducing the risk of serious thermal injuries (Hulka et al., 1987).

With the exception of conventional laparotomy, all surgical approaches and methods of tubal occlusion can be safely and effectively performed using local anesthesia. Because of the hazards inherent in the use of general anesthesia, the World Federation of Health Agencies for the Advancement of Voluntary Surgical Contraception (1984) advocates using local anesthesia for uncomplicated procedures.

A pregnancy that is identified after sterilization may have occurred because of conception before sterilization or because the operation was not successful. Conception before sterilization, called a luteal-phase pregnancy, is estimated to occur in about 2 to 3 per 1,000 interval tubal sterilizations (Chi and Feldblum, 1981). The most effective means of reducing the risk of luteal-phase pregnancy is to perform the sterilization before the estimated date of ovulation (Grubb and Peterson, 1985).

Rates of true sterilization failure vary by method of tubal occlusion, surgical expertise, and other factors, such as whether a woman has tubal disease. Overall rates are estimated to be between 0.2 percent and 0.4 percent per year (Table 2). No studies currently available permit rigorous comparison of failure rates by method of tubal occlusion, though one such study with the potential to do so is in progress in the United States. Since 1978 a multicenter cohort study conducted by the Centers for Disease Control has enrolled approximately 12,000 women undergoing tubal sterilization. These women are currently in various stages of a planned 5-year follow-up. Final study results will not, however, be available until the early 1990s.

When sterilization fails, the relative likelihood of ectopic gestation appears to be increased. Further, the risk seems to vary by method of tubal occlusion, with highest risk of ectopic pregnancy following electrocoagulation (Bhiwandiwala et al., 1982). The absolute likelihood of ectopic pregnancy after sterilization is actually reduced, however, relative to the risk a woman would incur when not using any contraception or using an IUD (DeStefano et al., 1982).

Studies from around the world suggest that tubal sterilization is a remarkably safe surgical procedure; however, more women die from tubal sterilizations than men die from vasectomies. Case-fatality rates for tubal sterilization have been estimated in reports from Europe, the United States, India, and Bangladesh. A French survey of approximately 100,000 laparoscopic procedures (including those for purposes other than sterilization) estimated a case-fatality rate of 20 per 100,000 procedures (Mintz, 1977). A prospective study of laparoscopic procedures from the United Kingdom reported a case-fatality rate of 10 per 100,000 procedures (Chamberlain and Brown, 1978). The case-fatality rate of tubal sterilization in U.S. hospitals has been estimated to be approximately 4 per 100,000 procedures (Peterson et al., 1982a). In a report from India the case-fatality rate from postpartum tubal sterilization was 7 per 100,000 procedures (Indian Council of Medical Research, 1982). In two studies from Bangladesh the case-fatality rate was estimated to be between 12 and 19 per 100,000 procedures (Grimes et al., 1982a, 1982b).

Worldwide, most sterilization-attributable deaths are caused by complications related to use of anesthesia, although deaths have occurred from hemorrhage and thermal injury (Peterson et al., 1983). In the United States 11 of 29 women whose deaths were attributable to sterilization suffered complications of general anesthesia (Peterson et al., 1983). Other reports from the United Kingdom and France have also identified complications of general anesthesia as the leading cause of laparoscopic-associated, sterilization-attributable death (Mintz, 1977; Chamberlain and Brown, 1978). Even in countries where general anesthesia is not often used, complications of anesthesia are still a major cause of sterilization-attributable death (Grimes et al., 1982a; Ross et al., 1985).

Reports regarding nonfatal complications attributable to tubal sterilization have varied by study design, definition, and classification of complications. Although comparing complication rates between studies is difficult, such studies indicate that major morbidity associated with tubal sterilization is uncommon and that safety varies with surgical and anesthetic technique. One large multicenter study, which included data from 23 countries, reported a complication rate of 2.0 percent for the approximately 7,000 women undergoing laparoscopy with silastic band application (Mumford et al., 1980). This rate was approximately twice that experienced by the approximately 5,000 women undergoing minilaparotomy procedures (0.8 percent). The WHO has reported the only randomized multicenter, multinational study. In that study approximately 800 women had minilaparotomies, and approximately 800 underwent laparoscopy using electrocoagulation. Major complications occurred in 1.5 percent of women undergoing minilaparotomy and in 0.9 percent of women undergoing laparoscopy. Minor complications were reported in 11.6 percent and 6.0 percent, respectively (WHO, 1982a).

The WHO also reported on a multicenter study of postpartum sterilization in which major complications occurred in only 0.3 percent of the patients (WHO, 1982b). This rate is identical to that reported from a large multicenter study of postpartum minilaparotomy conducted in India (Indian Council of Medical Research, 1982). In that study major complication rates associated with minilaparotomy did not substantially vary between those minilaparotomy procedures performed postpartum, those performed in association with early pregnancy termination, or those performed as an interval procedure. By contrast, women undergoing interval laparoscopy had a complication rate of 1.2 percent, and women undergoing interval colpotomy (via a vaginal incision) had an overall complication rate of 5.7 percent.

In a U.S. multicenter prospective study of laparoscopic tubal sterilization, 1.7 percent of women experienced at least one of six standard intra-operative or postoperative complications (DeStefano et al., 1983a). The most frequent major complication (1.1 percent) was unintended major surgery. In some instances this unintended surgery occurred because of incidental pathology identified during laparoscopy or because of technical limitations of laparoscopy itself. Thus, not all complications were directly caused by the laparoscopic procedure per se. The risk of complications was increased twofold or more by the following factors: obesity, lung disease, diabetes mellitus, previous abdominal or pelvic surgery, and a history of PID.

Although the overall major complication rate attributable to tubal sterilization is less than 2 percent, such complications can result in serious injury or death. The likelihood of serious injury appears to vary somewhat by surgical approach and tubal occlusion technique. Most minilaparotomy complications are not serious, with minor hemorrhage, minor wound infections, and uterine perforations reported most frequently (Liskin et al., 1985). By contrast, laparoscopy is more likely to result in serious complications. While rare, perforation injuries to major blood vessels or the gastrointestinal tract can occur (Peterson et al., 1982b). Open laparoscopy should markedly reduce the risk of these complications. One comparative study assessing complications among 1,112 women undergoing open laparoscopy and 288 women undergoing conventional laparoscopy failed to demonstrate a difference in complication rates (Bhiwandiwala et al., 1985). However, to detect a difference between open and closed laparoscopy, an epidemiologic study would have to involve thousands of women to have sufficient study power to detect a difference.

Complication rates also may vary with respect to method of tubal occlusion. For example, mesosalpingeal hemorrhage occurs more often from silastic band application than from alternative techniques. Unipolar coagulation increases the risk of thermal bowel injury. Although this type of injury likely occurs in fewer than 1 per 1,000 cases, more than 100 nonfatal thermal bowel injuries and 3 deaths from the use of unipolar instruments have been reported in the United States (Peterson et al., 1981a).

Because of the number of women involved, any important long-term sequelae of tubal sterilization would have major impact. On balance, the existing literature has failed to demonstrate any important negative long-term health effects of tubal sterilization. Most concerns regarding potential long-term sequelae have dealt with four issues: (1) menstrual abnormalities, (2) hysterectomy following tubal sterilization, (3) regret, and (4) ectopic pregnancy. We addressed the last concern previously in the section on sterilization efficacy.

Before 1980 most studies found an increase in menstrual abnormalities following tubal sterilization, the so-called post-tubal syndrome. Such studies, however, had major methodological limitations, including: (1) failure to identify and account for biases inherent in their retrospective design, (2) failure to use comparison groups, (3) failure to consider the effects of prior contraceptive use, (4) failure to consider women without menstrual changes or with improvements in menstrual symptoms, and (5) failure to evaluate information regarding presterilization menstrual patterns. Since 1980 most reports have had fewer methodological limitations and have failed to support the existence of a post-tubal syndrome.

One study has reported data from a series of approximately 10,000 women undergoing tubal sterilization at 64 institutions in 27 countries (Bhiwandiwala et al., 1983). Six menstrual symptoms were evaluated before and after sterilization, with most women reporting no changes 1 year after sterilization. When menstrual changes did occur, about as many women experienced improvement in symptoms as experienced deleterious change. A substantial percentage of the observed changes were attributable to cessation of OC or IUD use rather than to the sterilization procedure per se. No significant differences in menstrual patterns after sterilization were found among the various tubal occlusion techniques reviewed.

Another report concerned a sample of approximately 1,500 women selected from 45 centers in 24 countries (Fortney et al., 1983). The most important determinants of menstrual patterns after sterilization were the patterns at the time of sterilization. Women with abnormal patterns before sterilization were three times more likely to experience change than women with normal cycles; many of these women experienced change toward more normal cycles.

Several other reports from the United States and the United Kingdom that assessed menstrual patterns 1 or 2 years after sterilization failed to find support for the existence of a post-tubal syndrome. In the United States the Centers for Disease Control used data from a multicenter prospective study in which approximately 2,500 women were followed for 2 years after sterilization (DeStefano et al., 1983b). Each woman served as her own control; her menstrual function at the follow-up interview was compared with her menstrual function at the preoperative interview. Except for menstrual pain among women who underwent unipolar electrocoagulation, the prevalence of adverse menstrual function after tubal sterilization did not increase. Further, approximately 50 percent of women with adverse menstrual function preoperatively had an improvement by 2 years after tubal sterilization.

In a report from England 551 women undergoing interval sterilization were studied (Foulkes and Chamberlain, 1985). No increase in menstrual abnormalities among sterilized women compared with nonsterilized women was found after a year of follow-up. Both sterilized and nonsterilized women were more likely to experience heavy and prolonged menstrual bleeding if they had been using OCs before sterilization. Thus, discontinuation of OCs was more likely to result in abnormalities of menstruation than was sterilization per se.

Two studies, one from the United Kingdom (Vessey et al., 1983) and one from the United States (DeStefano et al., 1985), reported on menstrual function among women more than 2 years after their sterilizations. The study from the United Kingdom compared the experience of 2,243 women undergoing tubal sterilization with 3,551 women whose husbands underwent vasectomy. Three years after sterilization hospital referral or hospitalization for menstrual complaints was nearly equal for women undergoing sterilization and for the comparison group. After 6 years, sterilized women had a slight, but not statistically significant, increased risk for hospital referral or admission for menstrual complaints.

The study from the United States was principally designed to assess the long-term impact of OC use (DeStefano et al., 1985). In that analysis 719 women who underwent tubal sterilization were compared with 1,083 women whose partners underwent vasectomy. At follow-up, with intervals longer than 2 years, the tubal sterilization group had a significantly increased risk of abnormal menstrual cycles and combinations of two or more adverse menstrual outcomes. The likelihood of menstrual abnormalities was greatest, however, among the group of sterilized women who had presterilization menstrual complaints.

To date, no study has been reported that includes long-term follow-up for sterilized and nonsterilized women in a manner that allows comparison of specific menstrual symptoms and specific methods of tubal occlusion. The Centers for Disease Control in the United States is conducting such a study, but it will not be completed until the early 1990s.

Whether tubal sterilization increases the risk of subsequent hysterectomy is uncertain. Concerns that it does have been based, in part, on the possible existence of the post-tubal syndrome we noted earlier. Whether or not a post-tubal syndrome exists, the perception that it does may increase the likelihood that menstrual abnormalities after sterilization are managed surgically. Further, once a woman has been sterilized, either she or her physician may resort more quickly to surgical management of any gynecologic problem. These possibilities were assessed in the previously mentioned cohort study from the United Kingdom (Vessey et al., 1983). After both 3 years and 6 years of follow-up, hospital referrals leading to hysterectomy were equally likely in women who had undergone tubal sterilization and in wives of men who had undergone vasectomy. In Canada a population-based study using data from a health insurance plan studied 4,374 women aged 25 to 44 who underwent tubal ligation in 1974 (Cohen, 1987). As a comparison group, a random sample of 10,000 women registered in the insurance plan in 1974 was chosen. After 2 years of follow-up, no difference was found in adverse gynecologic outcomes between the two groups. However, beginning with the 2-year follow-up and increasing for up to 9 years, women ages 25 to 29 undergoing tubal sterilization had a statistically significant 60 percent increase in the probability of subsequent hysterectomy, even after controlling for other risk factors. For women aged 30 and over, tubal sterilization did not increase the likelihood of subsequent hysterectomy. The results from the two cohort studies with longer-term follow-up argue for continuing surveillance of sterilization sequelae beyond a 1- or 2-year period.

Published reports indicate that most women are satisfied with their decision to undergo sterilization. An overview of recent worldwide studies indicates that 2 percent to 13 percent of women interviewed 6 months to 6 years postoperatively expressed regret at having been sterilized; only 1 percent to 3 percent of those women seriously considered or underwent sterilization reversal procedures (Liskin et al., 1985). However, the prevalence of regret is likely to vary substantially by definition of regret. One survey of U.S. women found that 26 percent responded affirmatively when asked, "If it were possible for you to have another baby, would you, yourself, like to have one?" (Henshaw and Singh, 1986). In that same survey 10 percent of women responded affirmatively when asked, "As things look to you just now, if the operation could be safely reversed, that is changed back, would you want to have it reversed?"

Careful presterilization counseling is likely to minimize the chance of poststerilization regret. To maximize effectiveness, such counseling should focus on the reasons women might regret having been sterilized. A variety of studies have indicated that younger women, usually reported as less than 30 years of age, are more likely to regret having been sterilized (Grubb et al., 1985). In many developed countries divorce and remarriage are common reasons women regret sterilization and request its reversal.

A multicenter prospective study conducted by the Centers for Disease Control in the United States has found that only 2 percent of women regretted having a tubal sterilization 1 year after the procedure, compared with 2.7 percent after 2 years (Grubb et al., 1985). Five years after the procedure approximately 4 percent regretted it (unpublished data). Because the possibility for major life changes (e.g., death of a spouse or child) increases as women age, the longer the time since the procedure, the more likely women may be to regret having been sterilized. Thus, length of follow-up must be considered when comparing rates of regret in various studies.

In another study conducted in the United States, Shain et al. (1986) compared the likelihood that women undergoing tubal sterilization will regret it relative to the likelihood that women whose husbands underwent vasectomy will have regrets. At 1-year follow-up, only 2 of 234 (0.9 percent) women who had undergone tubal ligation compared with 2 of 154 (1.3 percent) women whose husbands underwent vasectomy expressed regret at having had the procedure. As the authors indicated, however, dissatisfaction with sterilization may involve various degrees of intensity, ranging from occasional concerns to desire for reversal. Further, these feelings are not static. When the authors used a broader measure of satisfaction/dissatisfaction as an outcome, women who had undergone tubal sterilization expressed significantly more positive feelings during follow-up interviews than did women whose husbands underwent vasectomies. This was attributed to a disproportionate prevalence of female control over reproductive decision making among women undergoing tubal sterilization. The authors concluded that counseling of the couple and the ability to exercise control over one's body are important predictors of sterilization regret.

Sterilization Reversal

The proportion of sterilized women who seek reversal is small but nontrivial (Ross et al., 1985). Highly sophisticated microsurgical techniques for tubal reanastomosis that increase the likelihood of successful reversal have been developed. These techniques are generally limited to developed countries. When available, such procedures are expensive and require laparotomy under general anesthesia. The likelihood of successful reversal depends on the method of tubal occlusion originally used. In general, 50 percent to 70 percent of women undergoing reversal surgery achieve intrauterine pregnancy (Liskin et al., 1985). However, most reports are from the selected series of highly skilled surgeons using sophisticated microsurgical techniques.

VASECTOMY

Vasectomy is cutting and occluding the vas deferens to prevent sperm transport in semen. Although generally considered to be safe, simple, and highly effective, vasectomy is not widely popular in most of the world. In fact, the majority of the estimated 41 million men currently using vasectomy for contraception reside in four countries—the United States, United Kingdom, China, and India (Gallen et al., 1986). By comparison, more than 95 million women worldwide have undergone tubal sterilization. Among regions, Africa is the least active in providing vasectomy services, Asia the most active, and Latin America moderately active (Ross and Huber, 1983). The limited popularity of vasectomy has been attributed to motivational factors and access to services (Gallen et al., 1986). In addition, female sterilization has become more popular than male sterilization in some countries (Ross and Huber, 1983).

Vasectomy is a minor surgical procedure that usually takes 5 to 10 minutes to perform (Ross et al., 1985). The procedure consists of isolating the vas deferens and then using one of three methods to occlude it: ligation, coagulation, or clip application. Ligation is the most widely used approach. If ligatures are tied too snugly, resulting in vas transection, or too loosely, resulting in incomplete occlusion, sperm can leak into surrounding tissues, resulting in sperm granuloma formation. Coagulation of the vas mucosa is an alternative method of occlusion that minimizes damage to the vas muscularis. Some believe that this method reduces the likelihood of sperm granuloma formation. Clips are rarely used for vas occlusion. Several clips must be applied to achieve maximum effectiveness, and, because they are not absorbable, the vasectomized man can sometimes feel the clips.

Local anesthesia without premedication is the anesthetic technique of choice for most vasectomies (Association for Voluntary Sterilization, 1983). The strong preference for local anesthesia is based on the higher risk of morbidity and mortality associated with general anesthesia (Peterson et al., 1981b, 1983). A survey of selected outpatient facilities conducted in the United States in 1980 indicated that the type of anesthesia used varied substantially by facility type (Kendrick et al., 1985). Freestanding surgical facilities reported performing 29 percent of their vasectomies using general anesthesia; conversely, 96 percent of the facilities not classified as freestanding reported using local or regional anesthesia. Outside the United States, particularly in developing countries, almost all vasectomies are performed using local anesthesia (Ross et al., 1985).

Vasectomy failure rates have ranged from 0 percent to 2.2 percent, with most studies reporting fewer than 1 percent (Liskin et al., 1983) (Table 2). However, few data are available that allow for a comparative assessment of failure rates associated with the various methods of vas occlusion. Most vasectomy failures can be attributed to unprotected coitus shortly after vasectomy or spontaneous reanastomosis of the vas. Unprotected coitus before sperm has been cleared from the male reproductive tract can result in pregnancy, although the duration of male fertility following vas occlusion remains uncertain (Lewis et al., 1984). Sperm may be absent within 1 to 10 weeks or more after vasectomy, depending in part on the frequency of ejaculation (Liskin et al., 1983). The Association for Voluntary Sterilization recommends using contraceptives for the first 15 ejaculations or 6 weeks after vasectomy (Ross et al., 1985).

Most true vasectomy failures occur when fistulous tracks develop through a sperm granuloma, resulting in a spontaneous reanastomosis of the vas (Liskin et al., 1983). The reanastomosis usually occurs within several months after vasectomy, although late reanastomosis up to 3 years after vasectomy has been documented, both after ligation and coagulation methods. In a study of 14,047 men the wives of 6 men became pregnant between 16 months and 3 years after vasectomy (Philp et al., 1984). Other causes of vasectomy failure include operating on the wrong structure and congenital duplication of the vas (Liskin et al., 1983).

Mortality

The risk of death attributable to vasectomy is quite low. A detailed review of sterilization-attributable deaths in Bangladesh occurring in 1979 and 1980 identified seven deaths attributable to vasectomy, for a vasectomy-attributable mortality rate of 31 per 100,000 procedures (Grimes et al., 1982a). Scrotal infection and sepsis caused most of the deaths. Subsequently, the use of sterile gloves during all vasectomies was recommended. Follow-up surveillance in 1981 identified no vasectomy-attributable deaths among approximately 14,000 men (Grimes et al., 1982b). The Association for Voluntary Sterilization has recorded only two vasectomy-attributable deaths associated with more than 160,000 procedures performed in the international programs it has supported (Ross et al., 1985). Vasectomy-attributable deaths in the United States are quite rare. While there has been no systematic effort to identify such deaths in the United States, at least one vasectomy-attributable death is known to have occurred (District Court of Appeals of Florida, Dunn v. Campbell, 1964).

Morbidity

The most common complications of vasectomy are swelling of the scrotal tissue, bruising, and pain. As many as 50 percent of men undergoing the procedure may experience these complaints, which generally subside within 1 to 2 weeks without treatment (Liskin et al., 1983). Hematoma formation and infection, which can be serious but generally are not, occur much less frequently.

Hematoma formation generally occurs in fewer than 1 percent of vasectomized men, but it has been reported in as many as 4 percent. Physicians surveyed in the United States in 1983 reported a hematoma rate of 2 percent (Kendrick et al., 1987). In that survey the hematoma rate was significantly higher among physicians performing 1 to 10 vasectomies per year (4.6 percent) than among those performing 11 to 50 vasectomies (2.4 percent) or more than 50 vasectomies (1.6 percent).

Infection after vasectomy is uncommon, usually occurring in fewer than 2 percent of men, although rates of infection as high as 6 percent have been reported (Liskin et al., 1983). Most infections occur at the site of skin incisions or around skin sutures. In rare instances, such as those reported in Bangladesh, infection can result in sepsis and death (Grimes et al., 1982a). Careful aseptic technique and sterile gloves are likely to reduce the risk of infection (Association for Voluntary Sterilization, 1983).

Epididymitis, manifested as swelling and tenderness of the epididymis, is generally reported in fewer than 1 percent of men undergoing vasectomy (Liskin et al., 1983). This complication has been attributed to a hydrostatic pressure increase within the epididymis after vas occlusion (Association for Voluntary Sterilization, 1983). Bacterial infection as an etiology of the epididymitis is uncommon (Schmidt, 1975). Epididymitis may develop several months after vasectomy, but it is most likely to occur soon after the procedure. Symptoms usually subside within a week (Ross et al., 1985). One report (Massey et al., 1984) compared vasectomized and nonvasectomized men with respect to the likelihood of developing epididymitis. After vasectomy, 2.6 percent of men experienced epididymitis versus 1.1 percent of men who had not had the procedure. However, differences between the two groups in the rate of epididymitis occurred only within the first year after vasectomy.

Sperm granulomas—nodules containing sperm, epithelial cells, and lymphocytes—represent an inflammatory response to sperm leaking into surrounding tissue. Most such granulomas are small and clinically not important, but they can result in pain at the site of vas occlusion or in the epididymis. The incidence of sperm granulomas is not known. However, granulomas at the vasectomy site have been found in 15 percent to 40 percent of sterilization reversal procedures; granulomas in the epididymis have been found in 10 percent to 50 percent of these procedures (Liskin et al., 1983). Symptomatic sperm granulomas are substantially less frequent. Follow-up studies report symptomatic granulomas in only 0 percent to 3 percent of vasectomies (Ross et al., 1985). In such instances a tender nodule at the site of vas occlusion or in the epididymis may cause discomfort, although the discomfort usually subsides spontaneously (Schmidt and Morris, 1973). As already noted, sperm granulomas can cause vasectomy failure if fistulous tracks develop through the granuloma. Ligation for vas occlusion is more likely to result in sperm granuloma formation than use of coagulation (Schmidt and Free, 1978). However, coagulation results in more complete occlusion of the vas, and this occlusion may increase the likelihood of an epididymal granuloma with resultant epididymal obstruction (Silber, 1978).

Long-Term Safety

A series of major epidemiologic investigations have been conducted in the 1980s to assess the long-term safety of vasectomy. In general, those studies are remarkably consistent in failing to identify any long-term health risks attributable to vasectomy.

Studies of long-term complications associated with vasectomy were stimulated primarily by two concerns: (1) evidence that 50 percent to 70 percent of men develop antisperm antibodies following vasectomy (Ansbacher, 1971; Schulman et al., 1972) and (2) reports of increased atherosclerosis in cynomolgus monkeys after vasectomy compared with nonvasectomized monkeys (Alexander et al., 1974). These findings are the basis for the hypothesis that antisperm antibodies form circulating immune complexes that might collect in arterial walls and contribute to the development of atherosclerosis (Alexander and Anderson, 1979). At least six epidemiologic studies specifically designed to assess this hypothesis have since indicated that vasectomy does not increase the incidence of MI in the 10 years following vasectomy (Goldacre et al., 1978, 1979; Walker et al., 1981; Petitti et al., 1982; Massey et al., 1984; Perrin et al., 1984). Subsequent to these reports, Rosenberg et al. (1986) evaluated data from a hospital-based, case-control study to assess whether vasectomy was associated with a subsequent increase in the risk of MI 10 or more years after surgery and whether any effect was more pronounced in men already predisposed to MI. The analysis supported findings from previous reports indicating that vasectomy did not increase the risk of MI within 10 years after surgery. Further, the authors found no overall increase in risk 10 or more years after surgery and no additional risk among men already predisposed to MI because of the presence of other risk factors for coronary artery disease. In summary, the available epidemiologic studies provide strong evidence that vasectomy does not increase the likelihood of MI.

As noted, much of the concern regarding the relationship between vasectomy and atherosclerosis was precipitated by reports of worsening atherosclerosis among vasectomized monkeys. Recently, the investigators who first reported this finding have presented data that do not support their first report. Their new data suggest that vasectomy does not increase the extent of atherosclerosis among cynomolgus monkeys, even in those found to be hyperresponsive to an atherogenic diet (Clarkson et al., 1988).

Several epidemiologic studies have assessed the relationship between vasectomy and a variety of other diseases associated with alterations of the immune system. In a historical cohort study conducted in the United States, 10,590 vasectomized men from four cities were interviewed, along with a neighborhood control for each vasectomized man (Massey et al., 1984). Any report of disease potentially related to vasectomy via an immunopathologic mechanism was validated by medical record review. Except for epididymitis/orchitis, the incidence of diseases, including those with a known or suspected immunopathologic basis, was similar for vasectomized men and paired controls. This finding was true not only for individual diseases but also for broader categories of diseases in which immunopathologic mechanisms might operate. Two other studies have failed to show an increase in the incidence of hospitalized illness for vasectomized men compared with nonvasectomized men (Walker et al., 1981; Petitti et al., 1982). The sole exception was that in one of the studies vasectomized men had higher rates of hospitalization for diseases of the genito-urinary system during the early postvasectomy period (Walker et al., 1981).

One recent report (Kronmal et al., 1988) raises additional concern regarding the relationship between vasectomy and subsequent genito-urinary tract disease by suggesting that vasectomy may increase the likelihood of urolithiasis. The age-adjusted relative risk for calculi among men who had undergone vasectomy was statistically significantly increased by approximately 70 percent. Although based on a study designed to evaluate the relationship between vasectomy and coronary artery disease, these findings are supported by another report mentioned previously (Walker et al., 1981). By contrast, another study found that the incidence of hospitalization for genito-urinary diseases was not significantly different between vasectomized and nonvasectomized men (Petitti et al., 1983). One possible biologic explanation for a relationship between vasectomy and urolithiasis centers around the production of antisperm antibodies. Whether antibody production might increase the tendency for formation of urinary calculi is not known. The possibility that vasectomized men may have an increased risk for genito-urinary disease warrants further evaluation, but available data are generally reassuring.

Vasectomy Reversal

Changes in life situation may lead men to regret their decision to undergo vasectomy. Surveys indicate that the prevalence of such regret worldwide is low (Liskin et al., 1983). Vasectomy reversal requires a high level of surgical expertise, is expensive, and is relatively unavailable in many parts of the world. Microsurgical techniques are necessary for optimal results. Furthermore, the original vasectomy technique may affect the likelihood of success of any subsequent reversal attempt. Because of the wide variety of factors influencing the likelihood of successful reversal, the success rate in the aggregate is difficult to estimate. Most surgeons report 30 percent to 60 percent success rates for vasectomy reversal, whereas some report even higher rates (Liskin et al., 1983). Even if the theoretical reversal rate remains consistently high, the technical difficulty of the surgery, with the attendant requirements for skilled microsurgeons, makes reversal impractical or unavailable for men in much of the world (Ross et al., 1985).

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Mechanisms for the Association of Maternal Age, Parity, and Birth Spacing With Infant Health

John G. Haaga

INTRODUCTION

Overview

This paper reviews possible biomedical mechanisms connecting maternal age, parity, and birth spacing with infant mortality and assesses their likely importance in accounting for the strength of these relationships as observed in household surveys in developing countries. This is partly to help refine estimates of the effects of family planning programs on infant health and partly to identify promising leads for research and the design and targeting of future maternal and child health interventions.

In a review of studies using data from the World Fertility Survey (WFS), Samuel Preston termed maternal age, parity, and birth spacing the "bio-demographic variables" affecting the risk of infant mortality (Preston, 1985, p. 265). Older studies using survey data and, to a lesser extent, data from vital events registration systems had shown excess risks for infants born to very young and very old mothers, for first births and for infants at very high birth orders, and for infants born after (and sometimes before) very short interpregnancy intervals. In the last decade or so analyses of demographic data, notably the WFS and similar retrospective surveys in developing countries, using methods designed to over-

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come at least some of the methodological problems, have brought these risk factors into new prominence. "Certainly the most surprising and probably the most important new finding from the WFS," as Preston (1985) puts it, "concerns the exceptionally high mortality rates among children born after a short birth interval" (p. 266).

Family planning programs can affect the distribution of all three of these risk factors in the population and may thereby be an important tool in reducing infant mortality rates. But the causal chains connecting family planning with the ultimate outcome of life or death for an infant are long and complex. "Young mother," "high parity," and "short interval" are not proximate causes of death such as would be listed on a death certificate. Insofar as the associations observed in demographic data are causal and not simply the result of confounding with other social or biological factors, it must be because the biodemographic factors increase the prevalence or severity of other conditions that are the more proximate causes of death. This paper reviews the evidence for some of the more likely mechanisms.

Even in low-mortality countries the large population-based studies that have provided most of our knowledge about the risks associated with these maternal factors (e.g., the Collaborative Perinatal Survey in the United States and the British Perinatal Study) have not been analyzed in a way that would show which direct causes of death are associated with the higher mortality rates for children born to very young and very old mothers and those born at high parity and after very short intervals. The WFS did not collect any information on causes of death.[1] So for this paper most of the evidence about direct causes of death comes from small clinical studies or special-purpose population studies, and their results do not readily or entirely explain the mortality differentials associated with the indirect factors that were measured in the demographic surveys.

None of the causal chains linking maternal characteristics with more direct risk factors and ultimately to poor pregnancy outcomes and infant deaths is entirely understood at the organ system or cellular level. For example, one can observe relationships between anemia or certain maternal infections and premature onset of labor, but it is not known what triggers labor or exactly how these maternal conditions affect the process. And the associations for which the evidence is strongest and the relative risks highest (notably the link between older maternal age and chromosomal abnormalities) are less important as causes of infant death in poor countries than in rich ones. A large part of the work for this paper consisted of culling results from obstetric, biological, and epidemiologic studies in rich countries and guessing their public health significance for poor countries.

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[1] The Demographic and Health Surveys are collecting retrospective cause-of-death information in one country on an experimental basis.

There have been a few good longitudinal field studies following infants from before birth in developing countries. But the logistical problems of maintaining a study group and obtaining accurate measurements and cause-of-death reporting preclude large samples. There are some interesting hospital-based studies, but these are always subject to severe selection and ascertainment biases in environments where most mothers and children do not get adequate care.

The remainder of this section discusses in more detail the differences between high- and low-mortality countries in the causes of infant deaths, which have a large influence on my subjective estimates of the likely importance of the causal mechanisms outlined in the rest of the paper. Next I list the possible causal mechanisms for which there is some evidence of association with young maternal age and primiparity. Then I discuss mechanisms that may link old maternal age and high parity with infant health. Finally, possible mechanisms for the effect of short interpregnancy or interbirth intervals on infant health are discussed.

To avoid repetition, maternal nutrition and infectious diseases are discussed only briefly in the second and third sections, which deal with maternal age and parity. They are dealt with more fully in the fourth section, on the effects of short intervals between pregnancies, for which their relevance is judged to be greater.

Causes of Infant Deaths

The pregnancy conditions for which evidence of an association with biodemographic variables is strong are not usually very important as causes of infant deaths in poor countries. Conversely, the major proximate causes of infant deaths in poor countries are not clearly related to biodemographic variables.

The causes of infant deaths differ between high- and low-mortality environments. It is difficult to obtain accurate information on causes of death when, as in most high-mortality countries, the majority of deaths are not attended by trained health workers. Even in low-mortality countries where most deaths occur in hospitals and there are strict requirements for certification, recording of causes can be unreliable and it can be difficult even to decide what is meant by cause of death in most situations, if a single cause must be chosen and recorded. But there is a reasonably clear picture of differences between high- and low-mortality countries, at least when causes of death are grouped into very broad categories.

Table 1 presents data on the causes of infant deaths in two reasonably well documented cases, corresponding to high- and low-mortality environments: (1) Recife, Brazil, in the late 1960s, when a special effort was made as part of the Inter-American Investigation of Mortality in Childhood to notify medical authorities and gather information on infant deaths as soon as possible after their occurrence and (2) the United States in 1983. Infectious and parasitic diseases account for a much larger proportion of the total when infant mortality rates are high. The lower the overall infant mortality rate is, the higher the proportion of

TABLE 1 Percentage of Infant Deaths by Cause: Recife, Brazil, and United States

	Recife, Brazil 1968-1971[a]	United States 1983
Infectious and parasitic diseases	51	2
Diseases of the respiratory system	11	4
Congential anomalies	4	21
Certain perinatal conditions	26	47
Ill defined	8	14
All other	—	11
Total	100	100
Infant mortality rate per 1,000 live births	91	11

[a] "Basic causes" only (from Inter-American Investigation of Mortality in Childhood).

Sources: Puffer and Serrano (1973), WHO (1987).

deaths attributable to congenital anomalies and to "certain conditions originating in the perinatal period," including birth trauma, hypoxia, and "immaturity."[2] And of the deaths due to these conditions, it is likely that a higher proportion of those in poor countries are attributable to poor obstetric care than to the age- and parity-related causes predominant in rich countries. For example, in the Narangwal study in the Punjab, 9 of the 24 deaths ascribed to "intrauterine asphyxia" were associated with administration of pytocin by indigenous practitioners to speed up labor (Kielmann and Associates, 1983, p. 197).

This contrast is important in the present context because it indicates the relative size of the effects that would have to be present for a particular category of cause of death to account for the observed indirect relationships between biodemographic variables and infant mortality. For example, if the effect of a short birth interval is associated with a relative mortality risk of 1.5 (relative to standard risk of 1.0 for births occurring after intervals of 2 to 4 years), and if this association is entirely causal and operates through only one of the categories of cause shown in Table 1 (an artificial and unlikely assumption), then a short birth interval would have to raise the risk of death from infectious and parasitic diseases by a factor of about 2. But a short birth interval would have to raise the risk of death from congenital anomalies by a factor of about 50 to account for the whole indirect association between short intervals and infant mortality. This alone makes it more likely that increased susceptibility to infectious disease, rather than increased incidence of congenital anomalies, is the most important underlying cause for the associations between biodemographic variables and infant mortality.

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[2] The figures shown in Table 1 are percentages of deaths corresponding to very different levels of infant mortality, of course. The mortality rate due to congenital abnormalities in Recife (4 percent of 91 per 1,000, or just under 4 deaths per 1,000 births) was higher than the same rate in the United States (21 percent of 11 deaths per 1,000, or just over 2 deaths per 1,000 births).

Low Birthweight and Prematurity

Low birthweight is associated with a higher risk of infant mortality, morbidity, and developmental problems (IOM, 1985). Infants may be small because they were born at less than full term (before the thirty-seventh week of pregnancy, counted from last menstruation, is a commonly used cutoff) or because they were born at full term after poor growth during uterine life (intrauterine growth retardation, or IUGR). Different combinations of gestational age and weight at birth are associated with different levels of risk to the infant.

Low birthweight and prematurity have many causes, and it is likely that the risk posed by a given deficit varies according to what caused it. The sections that follow discuss the limited evidence relating various biodemographic factors to birthweight and prematurity.

In relative terms, prematurity[3] accounts for a larger proportion of low birthweight infants in rich countries than in poor countries, where many more infants born at term are low in weight (Villar and Belizan, 1982). The relative risk of infant mortality is much higher for the preterm than for the term-but-low-weight infant. This point was brought out very starkly by a recent study of hospital births in Mexico City and Santa Cruz, Bolivia: the risk of neonatal mortality for preterm babies (relative to babies born at term and weighing more than 2,500 grams) was 24 and 100, respectively, in the two sites, compared to relative risks of 4.0 and 3.3, respectively, for babies born at term but weighing less than 2,500 grams (Haas et al., 1987). But the etiology of prematurity is still largely a mystery (Alger and Pupkin, 1986). In an interesting exercise in apportioning the attributable risk of low birthweight and prematurity, and the relative risk in rich compared with poor countries, among different proximate determinants, Kramer (1987) found that only about a quarter of variation in prematurity could be "explained" and much of that was with relatively uninformative determinants such as "nonwhite race."

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[3] I use the term *prematurity* and the adjective *preterm* only to mean early labor and delivery, usually defined with a cutoff of 37 weeks' gestation, counted from last menstruation. In older literature *prematurity* was often used for any low birthweight.

YOUNG MATERNAL AGE AND PRIMIPARITY

Effects on Birthweight and Prematurity

In the United States infants born to young mothers are at increased risk of premature delivery, low birthweight, prematurity, and perinatal death. Table 2 shows the relative risks of (1) birthweight less than 2,500 grams and (2) 5-minute Apgar scores (a composite index, including several signs associated with prematurity) less than 7 by mother's age for infants born in the United States in 1983. It can be seen that the risks of poor pregnancy outcomes are higher for younger mothers and that they are especially high among mothers less than 15 years old.

In high-mortality countries as well, infants born to young mothers are often at a disadvantage. In five of the Latin American project sites for the Inter-American Investigation of Mortality in Early Childhood in 1967–1970, infants born to mothers less than 20 years old were between 1.3 and 1.9 times as likely to die in the neonatal period as those born to mothers aged 20 to 24, and their relative risk was about as large for postneonatal deaths as well (Puffer and Serrano, 1973, Table 147). This was entirely associated with age, not primiparity, since a more detailed analysis showed that within mother's age categories first-parity births had the *lowest* neonatal death rates of all parities in most of the sites (Puffer and Serrano, 1975).

A more common finding is that primiparity is a risk factor for poor pregnancy outcomes over and above the effects of maternal age. The Collaborative Perinatal Study in the United States found that average birthweights were lowest for first births for both black and white infants born in 15 university-affiliated medical

TABLE 2 Risks of Low Birthweight and Low 5-Minute Apgar Scores, by Age of Mother, United States, 1983

Mother's Age (years)	BW < 2,500 grams	5-minute Apgar < 7
< 15	14.5%	4.1%
15	12.0	3.2
16	10.7	3.0
17	10.0	2.8
18	9.4	2.6
19	8.4	2.4
20–24	7.0	2.0

Source: National Center for Health Statistics, Advance Report of Final Natality Statistics, 1983 (Monthly Vital Statistics Report, September 1985).

centers in the years 1959–1965 (Table 3). Perinatal death rates (late fetal deaths plus deaths in the first week of life) were higher for first pregnancies than for second ones, although they tended to increase thereafter with parity. Neonatal death rates tended to increase, although not necessarily monotonically, with parity (Niswander and Gordon, 1972).

By contrast, in data from developing countries, first births do not consistently appear at a disadvantage. In a review of results of the WFS, Preston (1985) found the evidence for an excess mortality risk for first births equivocal. He suggested that in retrospective surveys women may tend to underreport first births of children who later died. The Inter-American Investigation of Mortality in Childhood, in which data were collected shortly after the time of death (and thus presumably would have been less subject to the type of recall bias Preston suggests), found that first births had the lowest mortality rates, within categories of mother's age, in the four project sites for which data were disaggregated—Chile, Mexico, Canada, and the United States (Puffer and Serrano, 1973, Table 151).

Mojarro and Aznar (1986) present infant mortality rates from rural Mexico by mother's age and parity (Table 4). They show that the excess risk for infants born to mothers less than 20 years old was for the second and third infants. Presumably a relatively high proportion of these babies are born after a relatively short birth interval and/or after a short gestation (a woman who gives birth for the first time at age 16 does not have enough time to have her third child before age 20, if she is to have the low-risk 2-year interval between births). In these data, infant mortality was actually higher for first-parity births to mothers aged 20 to 24 than for mothers less than 20. But the studies from rich countries showing the excess risk for the "older primipara" usually use the 20- to 24-year-old mothers as the refer-

TABLE 3 Birthweights and Perinatal and Neonatal Death Ratios by Parity and Race, United States, 1959-1965

	Mean Birthweight (grams)		Perinatal death ratios		Neonatal death rates	
Parity	Whites	Blacks	Whites	Blacks	Whites	Blacks
0	3,234	2,960	29	40	11	18
1	3,290	3,029	26	39	12	20
2	3,288	3,074	40	41	15	23
3	3,291	3,097	44	37	18	17
4	3,306	3,107	52	42	16	18
5+	3,340	3,138	61	51	20	23

Source: Niswander and Gordon (1972).

TABLE 4 Infant Mortality Rates by Parity and Mother's Age, Rural Mexico, 1965-1974

	Parity				
Mother's Age	First	2-3	4-6	7+	Total
< 20	69	106	[a]	[a]	86
20–24	108	65	101	[a]	83
25–29	[a]	56	75	84	72
30–34	[a]	[a]	75	79	77
35	[a]	[a]	99	100	101
Total	84	74	84	89	83

Note: n = 551 infant deaths, 6,640 live births.

[a] Fewer than 10 deaths in cell.

Source: Mojarro and Azner (1986, Table 8.3).

ence group with optimum conditions for childbearing. I suspect that the 20- to 24-year-old primiparae represented in the rural Mexican data include a large number of women who had prior miscarriages. The lowest infant mortality rates in this high-fertility population, in fact, are found along the diagonal of the table: these represent the women who are having the "right" number of live births for their age group.

Some of this difficulty could be removed if data were always available that separated the effects of parity (number of prior live births from gravidity (number of prior pregnancies), but fetal losses are underrepresented in both survey and certificate data, especially in high-mortality countries. The difference between the American and Mexican maternal age comparisons illustrates an important difficulty in making comparisons across populations of the effects of biodemographic variables on infant mortality. Since there are no random controlled trials (one cannot assign women to different parities at different ages), powerful social influences on the timing and pace of childbearing cause selectivity biases that are only partially and imperfectly controlled by the socioeconomic-status variables usually available. The biases vary among societies in magnitude and even direction.

It is difficult to separate in analyses the pure effects of young maternal age and primiparity from the effects on infant health of other factors associated with young motherhood and first parity in different societies. Especially in high-fertility countries, primiparous women are an increasingly select group in each older age group: women in their thirties giving birth for the first time have often had one or more previous pregnancies terminated by miscarriage or induced abortion. (A similar situation exists in sorting out the effects of old maternal age and "grand multiparity," though there is more variation at the end than at the beginning of reproductive careers, and the sample size, collinearity, and selectivity problems for statisticians are somewhat more tractable.) For the purposes of this review, the distinction between effects of young motherhood and effects of primiparity is quite important. Family planning programs can be expected to reduce the incidence of births to young women. But the proportion of all births in a population that are first births would likely increase as a result of widespread use of contraception to limit family size (Bongaarts, 1987).

Young maternal age is also confounded by environmental and possibly genetic characteristics that can affect infant health independently. In the United States, for example, young mothers are disproportionately black, poor, unmarried, and uneducated. They are less likely to receive antenatal care early in pregnancy than are older mothers, and the care they do get is often disjointed. Three recent reviews of the literature on the health consequences of adolescent pregnancy in the United States have all come to very similar conclusions (Strobino, 1987; Geronimus, 1987; McAnarney, 1987). As Strobino (1987) puts it, "adolescence, per se, may not be a risk factor for poor health outcomes of the mother or her offspring, but rather . . . the preponderance of other risk factors such as low socioeconomic status, poor prenatal care and primiparity is the reason for their poor outcomes" (p. 111).

To some extent this conclusion also holds for high-mortality countries, even though adolescent childbearing is not such a deviant behavior elsewhere as it is in the United States. Barros et al. (1987a, 1987b), for example, found higher rates of both perinatal and neonatal mortality among infants born to mothers less than 20 years old in a small city of southern Brazil. The mortality differences between these infants and those of mothers aged 20 to 29 completely disappeared when family income and whether the woman received antenatal care were controlled. DaVanzo et al. (1984), using recall data from a household survey in Malaysia, found that first births weighed significantly less than higher-order births. They found a positive bivariate association between mother's age (up to 35 years) and birthweight. The association between first-birth order and low birthweight persisted in a multivariate analysis controlling for family income and other social characteristics, while the association between young maternal age and low birthweight disappeared in the multivariate analysis. In an analysis of determinants of infant mortality using the same data, younger maternal age (below 18 years) was associated with increased risk of infant death, but the association was much diminished when social characteristics and behavioral variables (notably breastfeeding) were controlled (DaVanzo et al., 1983).

The results of these Brazilian and Malaysian analyses tend to support the conclusion that young maternal age is less important than primiparity as a risk factor for low birthweight and infant mortality. It may be that *very young* maternal (gynecologic) age poses great risks to the infant, but in the studies reviewed for this paper the age cutoffs used to define young maternal age were sufficiently high that the excess (relative) risk was small. Small sample sizes in population-based studies typically preclude estimation of infant mortality risks for very young mothers, which means that this potential risk factor is not a major attributable risk for infant mortality in the countries for which we have good information.

The remainder of this section examines in turn the major pathways or proximate causes through which young maternal age could be associated with lower birthweight and infant deaths.

Mother-Fetus Competition for Macronutrients

It is generally thought that girls are still growing for 4 years after menarche and that the energy cost of a pregnancy during these 4 years must compete with the energy cost of linear growth of the mother (NRC, 1970). In analyzing data from the Collaborative Perinatal Study, Naeye (1983) showed that infants born to very young (10- to 16-year-old) black mothers had low birthweights and lengths, which he suggested could be explained by the "fetal-maternal competition for nutrients." And in an analysis of data on infants of very young black mothers in New Orleans, Cherry et al. (1987) showed that this birthweight and length deficit persisted through infancy into childhood. But other studies have shown that for most girls the linear growth spurt in adolescence takes place well before menarche or during a period of adolescent subfecundity (see McAnarney, 1987, for a review). The energy cost of linear growth is a fairly small percentage of metabolic requirements even for young children, and linear growth is interrupted quickly when there is nutritional stress. In any case, as discussed further later in this paper, the energy costs of pregnancy are surprisingly low and can usually be met at the expense of maternal fat deposition even during nonharvesting seasons in very poor communities in West Africa.

On the face of it there appears to be little reason to think that absolute shortages of macronutrients are a binding constraint on fetal growth except in the most extreme circumstances. McAnarney (1987) suggests that the small size of the infants born to very young mothers in the United States is more likely due to the mother's small size at maturity, before pregnancy, and to their greater rates of infection and premature delivery than to a simple competition between two growing children, one in the womb of the other. It may also be that the problems are not so much with the quantity of nutrients in the mother's circulatory system as with the size and development of the uterine blood vessels early in pregnancy: a fetus can be undernourished even when there are "enough" nutrients around if the maternal-fetal transfer is impaired.[4]

The situation may be different in poor countries. Age at menarche occurs later on average in poor countries than in Northwest Europe and North America, where secular declines in age at menarche have been documented. At the same chronological age, teenagers in poor countries are usually at a younger gynecologic age than their contemporaries in rich countries. Growth may last longer after menarche as well.[5]

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[4] I discuss this point further below in discussing short spacing between pregnancies. This point has been made in connection with studies of placenta morphology and IUGR in general, but my connection of it to the specific problem of adolescent pregnancy does not have any other support in the literature.

My tentative conclusion is that the evidence is weak for an effect of young maternal age on fetal growth and infant health through direct competition for nutrients. More plausible is an association caused by the connection of both very young maternal age and poor infant health with poverty and poor sanitary conditions. There is little evidence directly bearing on the issue of young mothers, but it could also be that "uteroplacental perfusion" of nutrients is impaired in very young women, as it appears to be in animal studies of undernutrition in pregnancy.

Micronutrient Deficiencies

Iron and folate deficiencies are common in pregnancy, and routine supplementation is recommended by the World Health Organization (WHO). In the United States, teenage mothers are often found to be more anemic than older mothers, but this effect is most likely due to their poverty and poor diets rather than to any true age effect (Strobino, 1987). The more common finding in poor countries appears to be an association of iron and folate deficiencies in high-parity pregnancies (anemia is discussed in greater detail later in this paper.

Zinc is a micronutrient that has attracted a great deal of attention in recent research (Swanson and King, 1987). It is suspected that one of the ways in which maternal smoking and alcohol intake affect fetal growth is by interfering with the mother's zinc utilization. Whether because of these behaviors or because of dietary deficiencies, low circulating zinc levels have been found in pregnant teenagers in the United States. Pregnancy-induced hypertension and poor pregnancy outcomes (e.g., prematurity, IUGR, congenital malformations) have been associated in observational studies with low circulating zinc levels, but so far results of supplementation trials are equivocal. Apparently the assessment of zinc status is extremely difficult, and the studies reviewed by Swanson and King have all been conducted in low-mortality countries. For the purposes of this paper, zinc deficiency must be considered a factor that may prove an important link between biodemographic risk factors and infant health in developing countries, but there is virtually no evidence yet.

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[5] This is at least a possibility. It is known that children in poor communities, where undernutrition and infectious diseases are prevalent, are shorter than those of similar genetic background who have had less stress, at least until the age of 7 or so. But adults in poor countries (e.g., West Africa) are not much smaller than those from rich countries. It has been suggested that linear growth lasts longer (say, into the early twenties) to account for this discrepancy (Habicht et al., 1974). This might prolong the period between menarche and the end of linear growth.

Infections

Infections of the urinary tract are associated with premature delivery and neonatal sepsis.[6] I have found no studies showing differences by age and parity in maternal or fetal *susceptibility* to infections, which leaves differences in exposure as a likely mechanism. Young mothers may be more likely than older ones to have sexually transmitted diseases (STDs) and other genito-urinary infections, presumably because in many countries young mothers are a select group, with lower social and economic status and perhaps with more sexual partners than much older women.

Congenital syphilis was an important cause of perinatal mortality in one urban African sample (Naeye et al., 1977). Efiong and Banjoka (1975) found syphilis in 7 out of 95 primigravidae less than 16 years old in urban Nigeria, compared to none in 100 older primigravidae selected as controls. A prospective study in Sierra Leone found that pregnant women under age 20 were more likely to have both urinary and genital tract infections in pregnancy than were older pregnant women (WHO, Family Health Division, 1981).

Pregnancy-Induced Hypertension

Pregnancy-induced hypertension (PIH) is associated with premature delivery and placental abruption, both of which are associated with low birthweight (Niswander, 1977).[7] In the 1958 British Perinatal Mortality Study it was found that 18 percent of infants born to mothers diagnosed as having pre-eclampsia (PIH and edema) weighed less than 2,500 grams at birth, compared to 5.4 percent of the infants born to normotensive women, and that 8.5 percent of the deliveries following a diagnosis of pre-eclampsia took place before 37 weeks of gestation, compared to 5.1 percent of deliveries for the normotensive women (Alberman, 1984). Undetected PIH can progress to the convulsions of eclampsia, putting both the pregnant woman and her unborn child at very severe risk of death. Even among women diagnosed as having toxemia, or pre-eclampsia, who are under good obstetric care and who do not progress to eclampsia, the risk of perinatal mortality is elevated—double in one study in the United States in the 1970s (cited by Davies and Dunlop, 1983, p. 201).

The precise etiology of PIH is unknown, but it is thought to be a reaction of the maternal circulatory system to the placenta because the syndrome never appears in nonpregnant women and because it is so powerfully associated with first pregnancies. About 1 in 20 primigravidae in the United States are diagnosed as having PIH, and the risk of recurrence is fairly high (about a third of those who have it with one pregnancy will get it again); most of the multigravidae with PIH had it in their first pregnancy (Mehta and Young, 1987). The incidence is highest in the United States for poor black women.

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[6] Other maternal infections are discussed later in this paper.

[7] PIH is the term generally used now in North America. The more common term in Britain is *gestational hypertension*.

Unfortunately, PIH is not easy to diagnose, especially in settings where women do not get medical attention early and often during their pregnancies. Older women and black women in the United States have higher rates of underlying ("essential") hypertension, which poses little risk. Many women have a drop in blood pressure in early pregnancy followed by a rise back to their prepregnancy levels; this is often a less serious indication easily be mistaken for PIH (Davies and Dunlop, 1983). It is thus very difficult to estimate the prevalence of PIH in either low- or high-mortality countries.[8]

In an analysis of data from a referral hospital in Harare, Zimbabwe, Crowther (1986) had multiple blood pressure observations for enough of his subjects to distinguish between essential hypertension (he used the term *chronic hypertension*) and PIH, the latter being defined as high blood pressure appearing after the twentieth week of gestation in a patient previously normotensive or normotensive after the sixth week postpartum. He also used the presence or absence of proteinuria to diagnose pre-eclampsia (PIH plus proteinuria). About 45 percent of the PIH cases were women less than 20 years of age, compared to 20 percent of the general obstetric population. Among the hypertensives the diagnosis of PIH was more common among those less than 30 years old and the diagnosis of chronic hypertension more common among those over 30 years old. Perinatal mortality was much higher (79 per 1,000 live births) for the hypertensives than for the normotensives (26 per 1,000). Interestingly, the women in Crowther's sample with only PIH (no proteinuria) had perinatal mortality rates comparable to those of the normotensive women; the excess rate for hypertensives was due to higher death rates for infants born to women with pre-eclampsia and chronic hypertension. This underlines the heterogeneity of the "high blood pressure during pregnancy" population. A series of blood pressure readings is important for surveillance, which of course depends on regular antenatal care and good recordkeeping.

Harrison (1985) reports rates of obstetric complications for hospital births in northern Nigeria (Table 5). Among primigravidae the rates of pre-eclampsia were slightly higher for women below age 20, rising again for those over 25 (who accounted for only 7 percent of first births). There was a much starker age

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[8] Davies and Dunlop (1983) cite the experience of the Collaborative Perinatal Study in the United States to show that even "first-class departments of obstetrics give contradictory results even with agreed protocols and definitions" (p. 182).

TABLE 5 Rates of Pre-eclampsia and Eclampsia Among Singleton Hospital Births in Northern Nigeria, by Age and Parity

	Age of Mother						
	< 15	15	16	17–19	20–24	25–29	30+
Pre-eclampsia							
Primigravidae	11.6	10.4	10.3	10.2	9.9	15.0	12.9
Multigravidae				(20-29, parities1-4 = 3.4) (30+, parity 5+ = 5.8)			
Eclampsia							
Primigravidae	16.7	12.8	7.4	4.9	2.7	2.5	.32
Multigravidae				(20-29, parities 1-4 = 0.5) (30+, parity 5+ = 0.3)			

Source: Adapted from Harrison (1985, Appendix 4.3).

differential in the more serious diagnosis of eclampsia, however: the prevalence of pre-clampsia in primigravid women less than 15 years old was six times that for primigravid women aged 20 to 24. Ojo and Oronsaye (1988) present data from another hospital-based study in Nigeria, again showing higher rates of pre-eclampsia for younger primigravidae than for those in their twenties (33 percent for women less than 20 years old compared to 21 percent for those aged 20 to 24).

One of the main problems for maternal and child health in this population, according to Harrison (1985), is "the strongly held belief that home delivery is best for all primigravidae, especially the youngest" (p. 91), which may account for the very high rates of eclampsia relative to pre-eclampsia. Lawson and Stewart (1967) similarly observed that PIH is a much more threatening condition for women (and I presume for the infants if their mothers survive) in poor countries because they often present at hospitals only after convulsions have begun. The lack of adequate antenatal care in many places could lead to a detection bias that makes it difficult to interpret statistics based on hospital births: higher rates of pregnancy complications among young women may be due to differences among age groups in the likelihood of an uncomplicated delivery taking place in a hospital as well as to true differences in the prevalence of complications.

Malaria

Malaria is a major public health problem in the tropics. The WHO estimates that there are over 100 million clinical cases (new and recurring) each year. Half the world's population lives in areas covered by antimalarial campaigns, yet the overall situation has been "static" in recent years (WHO Malaria Action Programme, 1987).

Because of its prevalence, if malarial infestations affect differentially the health of infants born at different parities or to mothers at different ages, then even a fairly small effect could account for a large part of the excess risk attributable to maternal age or parity.

In regions where malaria is relatively rare and where many adult women do not have acquired immunity, or among migrants who did not acquire immunity as children, malaria during pregnancy is devastating for both mother and fetus or neonate. More significant for our purposes is the situation in the large regions of the world, notably in sub-Saharan Africa, where malaria is "holoendemic"; "Where indigenous adult women have acquired significant protective immunity, the effects of malaria on pregnancy and its outcome are much more difficult to assess" (McGregor, 1984, p. 517).

One theory is that there is a breakdown in acquired immunity during early pregnancy and that maternal immunity is only gradually reacquired in late pregnancy.[9] In any case the placenta appears to be a preferred site for the malaria plasmodia. Brabin (1983) reviewed recent hospital studies in Tanzania and Cote d'Ivoire in which about a third of all placentas were infested by *falciparum* (the most common agent of the infection during pregnancy in Africa). Parasitization of the placenta appears to interfere with the circulation of maternal blood in the spaces between villi, and it is associated with low birthweight, though it is still not clear how much of this effect is due to prematurity and how much to intrauterine growth retardation (Lawson and Stewart, 1967; Brabin, 1983). In 10 studies reviewed by Brabin, infants born to women whose placentas were infested with *falciparum* had birthweights that were on average 50 grams less than those of women whose placentas were free of the parasite.

It is suspected that placental malaria is also associated with greater risk of late fetal death and stillbirth, but "no study has quantified the part it plays in inducing spontaneous abortion, or established a clear and significant relationship between it and enhanced stillbirth rates" (McGregor, 1984, p. 523).

In African studies primigravidae have about double the rate of placental malaria of multigravidae. Bray and Anderson (1979) presented population-based data for 1,000 pregnancies in several Gambian villages, showing that 59 percent of primiparae were infected during pregnancy, compared with 31 percent of women at parity 4 or more. Neither the causes of the parity difference in infection rates nor its implications for stillbirth rates, infant growth, and infant mortality are clear. Nonetheless, McGregor (1984) reviewed the fragmentary evidence from

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[9] Judith Fortney (1988, Family Health International, personal communication) points out that maternal immunity to non-A non-B hepatitis, common in parts of Africa and South Asia, may similarly break down in pregnancy.

Africa showing that placental malaria infestation was not only more common in primigravidae but also that the associated maternal anemia and birthweight deficit were larger for primigravidae than multigravidae.

Apparently congenital malaria or febrile attacks in infancy are rare in areas where malaria is endemic (Lawson and Stewart, 1967; McGregor, 1984). The effect of malaria during pregnancy is likely to be most pronounced with respect to stillbirth rates; its effect on infant mortality would be through the increased susceptibility of the premature and low-birthweight infant to other infections. Bray and Anderson (1979) reviewed evidence that malarial infection interferes with the transmission of Immunoglobin G from the mother to the fetus, so that "the infant nourished from the infected intervillous spaces of the placenta may, three months later, be at a disadvantage to malaria parasites" (p. 429); due to depressed immunocompetence and lower birthweight, such infants may be more susceptible to bacterial or viral infections as well. I have found no studies that followed infants born to mothers with infested placentas and assessed their risks of postnatal morbidity and mortality.

The association of malaria in pregnancy with first parity is the only clear link between the biodemographic variables of interest here and parasites involving infants.

Complications of Delivery

Fortney et al. (1986) present evidence from records collected from 86 hospitals, mostly in developing countries, showing that in cases of vaginal delivery of a breech presentation, the perinatal deaths rates were significantly lower for multiparae than for primiparae. Obstetric management of breech presentation is probably more aggressive for primiparae, but of course many of the poorest women have no access to facilities that can perform a safe caesarean section. Thus breech presentation may be more common for grand multiparae, but higher case-mortality rates for primiparae would lead to a parity differential, probably small, in infant mortality.

Conclusions

The mechanisms through which young maternal age and primiparity can affect infant health and for which the evidence is strongest are pregnancy-induced hypertension, intrauterine growth retardation, and placental malaria. All three are associated with primiparity more strongly than with young maternal age per se. The evidence that a young mother's own linear growth requirements lessen nutrient availability for fetal growth is weak. The disadvantages associated with young motherhood are primarily those of poverty and ignorance, since young mothers in both rich and poor countries tend to include disproportionate numbers of the poorest and least educated women. Young mothers have been found, for example, in several studies to have higher rates of genital and urinary infections, which can cause prematurity.

There appears to be an interaction between age and primiparity such that first births to older women are at a particular disadvantage. Some of this effect is due to higher rates of chromosomal abnormalities for infants born to women nearing the end of their fertile years. Older primiparae in poor countries are usually a very select group, however, including many women who aborted previous pregnancies, so it is difficult to separate the effects of age from those of fecundity and other woman-specific factors not easily measured in the data available to demographers.

Even if one concludes that the associations between young maternal age and infant mortality are not true age effects, it may still be true that family planning programs could affect infant health by lowering the percentage of births to adolescents. Insofar as the programs reach poor and uneducated women and give them control over their fertility, they would allow women to postpone births until they were able to support more children.

PIH, many maternal infections, and malaria are all conditions that even "low-tech" antenatal care is designed to detect and/or treat. If family planning is introduced as part of a general extension of maternal and child health services, the excess risk associated with primiparity for most women in poor countries should be reduced.

It would be useful to compare the excess mortality risk for first births in regions of malaria endemicity with that in comparably poor but nonmalarious regions. In areas where first births appear to be at greatest disadvantage, more detailed field studies would be indicated. It would be impossible to trace the full course of the postulated mechanisms in a prospective study, since detectable maternal conditions would have to be treated. But field studies would usefully supplement current evidence, most of which is based on hospital data of uncertain selectivity, showing associations of young maternal age and primiparity with preventable diseases in pregnancy.

OLDER MATERNAL AGE AND GRAND MULTIPARITY

Relative Risks of Poor Pregnancy Outcomes and Infant Deaths

Hansen (1986) reviewed studies, mainly from low-mortality countries, of the effects of older maternal age on the health of mother and infant and concluded that "the literature seems to support the finding that older women have increased incidences of babies weighing less than 2,500 grams at birth (probably both preterm and small-for-gestational-age) and babies more than 4,000 grams. This is consistent with the finding of increased rates of hypertension and pre-eclampsia (preterm and SGA) and diabetes (large babies) among older pregnant women" (pp. 731–732). Diabetes is rare enough (and the infant mortality differential associated with very high birthweight is small enough) that it is unlikely to produce a discernible impact on infant mortality in poor countries. PIH, prematurity, and chromosomal abnormalities (for which infants born at first parity to older mothers are at increased risk) are the more important pathways for this review.

Besides older maternal age per se, grand multiparity has been found to be associated with infant mortality in many studies. Like very young maternal age, very high parity is often a "marker" for low social status and income, since fertility rates are inversely associated with income in societies that have begun the demographic transition. This and a more direct selection effect caused by purposeful efforts to replace children who have died complicate the interpretation of infant mortality rates at high parity.

The British Perinatal Mortality Survey of all pregnancy outcomes in a period during 1958 showed a U-shaped association of rates of both low-birthweight and preterm births with parity (Table 6).

Fortney et al. (1983), in comparing data on deliveries in urban teaching hospitals in Mexico and Egypt, found that the largest association of older maternal age and child survival was for first-parity births (Table 7).[10]

These mortality differences were not due to differences among mother's age groups in the prevalence of low birthweight; in fact, the older primigravidae had a lower incidence of low birthweight than either younger primigravidae or older multigravidae (Fortney et al., 1983, Table 4).

Similarly, in analyzing data for a "natural fertility" religious group in Israel, Seidman et al. (1987) found that stillbirths were more common for grand multiparae (parity 6 and over). This mortality differential was not associated with a parity effect on birthweight, since rates of low birthweight decreased monotonically with parity within sibship groups. The very highest parity (8+) infants in this study had higher rates of low birthweight than lower-parity infants, but Seidman et al. show that this difference is reversed when sibship groups are compared: the high-parity infants weigh more at birth than their older siblings did. They argue that this is evidence of a form of selection, presumably biomedical, since this is an aggressively noncontracepting population. That is, women who have small babies are more likely than others to progress to higher parities.

Bakketeig and Hoffman (1979) demonstrated similar effects using data on singleton births at parity 4 or less plus late fetal deaths in Norway. Perinatal mortality and rates of low birthweight were both larger for first parity outcomes in the marginals for the total sample. But within groups defined by the number of births to the same woman in a 6-year period, both rates fell monotonically with

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[10] I have omitted here the data from Hungary that Fortney et al. present, which did show an association between older maternal age and low birthweight but that also showed much higher survival rates in all groups and no association of maternal age with survival.

TABLE 6 Low Birthweight and Preterm Births by Parity, Britain, 1958

Parity	Birthweight < 2,500 grams	Gestation < 37 weeks
0	7.6%	4.7%
1	5.4%	3.9%
2–3	6.8%	5.2%
4+	7.4%	5.5%

Source: Alberman (1984).

TABLE 7 Percentage of Neonates Surviving Until Mother's Discharge from Hospital, by Mother's Age and Parity, Mexico and Egypt, 1977–1980

	Mexico		Egypt	
Mother's age	20-34	35+	20-34	35+
Parity				
0	96.7	89.2	96.0	87.5
1–3	96.6	95.6	95.3	90.5
4+	94.9	90.3	91.8	87.3

Source: Fortney et al. (1983).

parity. Only children were overrepresented in the first parity group, children from two-child families are overrepresented in the first two parities, and so forth. Bakketeig and Hoffman's results are hard to interpret because they pertain to births during a specified time period (1967–1973), so their large families may consist disproportionately of short-interval births. Also, the replacement effect—intentionally quick conception following a fetal or infant death—is not well controlled in this analysis (though its effects on birthweight should have been negligible).

In the Collaborative Perinatal Study in the United States, higher-parity infants were at much higher risk of death both in the perinatal and neonatal periods (though the neonatal mortality differential associated with parity was much smaller for blacks than for whites) (Niswander and Gordon, 1972; see Table 3 above).

In reviewing the literature mainly from low-mortality countries, Hansen (1986) concluded that "there is an association between age and risk for spontaneous abortion, but the magnitude of the risk is unclear because of the unknown contribution and/or study bias of the factors of gravidity, birth order, and reduced fecundity" (p. 735).

Interpretation of the cross-sectional associations of age and parity with spontaneous abortion and prematurity is difficult because of the high risk of repeat abortions (Mehta and Young, 1987). Especially in societies in which fertility control is efficient, women who are trying to have babies at older ages and higher parities may disproportionately consist of those who have had previous abortions and stillbirths. Powell-Griner (1987) presented results that distinguish neatly the effects of parity with and without prior pregnancy loss. The data set she used was created by linking birth and infant death records for all births and late fetal deaths (*n* = 647,953) in eight states in the United States in 1982. Table 8 shows the perinatal death rates for various combinations of parity, birthweight, and prior loss, crude and adjusted for effects of social/demographic variables and adequacy of prenatal care.

In both birthweight categories, perinatal death rates for infants of parities 4 and over were well below those for first-parity infants if no prior loss was reported and well above those for first-parity infants when there was a prior loss. Adjustments for maternal characteristics and adequacy of prenatal care made these differences even starker. For the "no loss" multiparae the adjusted perinatal death rates for parities 4 and over are slightly higher than for parities 2 and 3, though both are far lower than for first-parity infants in either weight category. For the "prior loss" multiparae both crude and adjusted rates are lower in each weight category for parities 4 and over than for parities 2 and 3. Parities 2 and 3 usually appear in the

TABLE 8 Perinatal Death Rates by Parity, Birthweight, and Prior Pregnancy Loss, United States, 1982

Parity/Prior Loss	Birthweight, 1,500-2,499 grams		Birthweight, 2,500-3,999 grams	
	Crude	Adjusted[a]	Crude	Adjusted[a]
First	39.4	41.1	3.7	4.6
2–3, no loss	15.4	1.4	1.1	0.1
4+, no loss	11.9	3.6	1.3	0.4
2–3, prior loss	94.7	137.0	8.6	10.1
4+, prior loss	49.7	63.6	6.2	8.0

[a] Adjusted for maternal age, race, education, marital status, and whether or not the mother received adequate prenatal care.

Source: Powell-Griner (1987).

literature to be the safest, so I can suggest only that this last difference is a selection effect: women in the parities 2 and 3 group with prior loss have had a very high proportion of their pregnancies end in fetal death. Those in parity 4 include many women with lower proportions of aborted pregnancies and presumably greater intrinsic ability to bring babies to term. Powell-Griner's (1987) results show that the excess risk of perinatal death for higher parities in the United States nearly disappears when prior loss is taken into account: the risk is very largely a risk of recurrence.[11]

Kiely et al. (1986) performed a very useful analysis of certificates for all singleton live births and late fetal deaths in New York City during 1976–1978, separating age and parity effects and mortality in the late fetal period, before and during labor, perinatal deaths attributable to congenital abnormalities, and neonatal deaths for causes other than congenital abnormalities. This is one of the few analyses from any country associating age and parity with cause of death. Kiely et al.'s multivariate analysis controlled for mother's race, educational attainment, and marital status. Fetal deaths were most strongly associated with maternal age but are less relevant for our purposes because they are theoretically unrelated to the infant mortality differentials we are trying to explain. For intrapartum deaths there was no risk difference by maternal age. Infants born to mothers over 35 were at increased risk for perinatal deaths attributable to congenital abnormalities (relative risk of 1.54 compared with infants born to women aged 25 to 29). For neonatal deaths due to other causes, there was no main effect of high parity nor was there an effect of maternal age for infants of second or higher parity. There was, however, a clear age differential in the risk of neonatal mortality for first-parity infants born to mothers of different ages. First-parity infants born to mothers aged 35 and over had a relative risk of 2.33 (95 percent confidence interval: 1.75–3.09) for neonatal death for causes other than congenital anomalies, compared with 1.43 (1.23–1.68) for first-parity infants born to mothers aged 25 to 29. (In each case the risk is compared with the risk to higher-parity infants born to women aged 25 to 29.) Kiely et al. suggest that better obstetric management of high-risk pregnancies has resulted in more infants of older primiparae surviving for a few days or weeks, albeit in a weakened state. This explanation would not hold for most women in high-mortality countries, who have no access to obstetric and neonatal intensive care.

In a prospective study in rural central Java, Indonesia, effects of maternal age and parity on child mortality at different ages were distinguished (Santow and

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[11] The "high-parity" category in this analysis is of course not very high by world standards. Analyses of data from the United States that use higher cutoffs run into very severe sample size problems (as it was, Powell-Griner used linked certificate data on nearly a quarter of all births in the United States that year) and the selectivity biases noted in the text. This makes "natural fertility" samples like those of Seidman et al. especially valuable.

Bracher, 1984). Primiparity was associated with greater risk of death in early infancy, suggesting prenatal influences, while older maternal age and high parity were associated with greater risk in later infancy and during the second year of life, suggesting causes associated with the postnatal environment. This study had small numbers of deaths, illustrating again the difficulty of combining good measurement of independent variables and realistic field conditions with large samples and statistical tests of high power.

Congenital Abnormalities

All the studies reviewed by Hansen (1986) found an association between older maternal age and chromosomal abnormalities in aborted or stillborn fetuses and congenital abnormalities in live-born infants. The association of advanced maternal age with Down's syndrome has been known for decades, and more recently it has been linked with other chromosomal abnormalities (Hook, 1985). Maternal age has a "strong, ubiquitous positive association with all viable trisomies [including trisomy 21, which causes Down's syndrome], and with most that are not viable," according to Hook (1985, p. 126). Some abnormalities appear to be associated with paternal age as well, but not as strongly as with maternal age. The explanation for the association of maternal age and chromosomal abnormalities is apparently unknown. It is usually ascribed to some biologic aging of the ova. Hook suggests that it may in part be an effect of higher rates of late fertilization of ova due to lower frequency of sexual intercourse among older people.[12]

Other malformations not attributed to chromosomal abnormalities (e.g., spina bifida, cleft palate) have also been found to be associated with advanced maternal age, but the age gradient is not so steep (and in the case of the more common condition, cleft palate, the case mortality rates are not so severe) as for chromosomal abnormalities (Hansen, 1986).

Iron-Deficiency Anemia and Micronutrient Deficiencies

The main way in which maternal anemia could affect infant health is through an association with premature delivery. Requirements for dietary iron increase greatly during pregnancy, approximately quadrupling in the last trimester when stores are laid down in the fetal liver. Pregnant women are often found to have lower hemoglobins than nonpregnant women (Royston, 1982). Anemia is particularly severe in areas where hookworm infestation is common, throughout the wet tropics (Lawson and Stewart, 1967).

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[12] Frequency of intercourse is usually related more strongly to duration of marriage than to age (with which it is confounded), but I found no studies reporting incidence of congenital abnormalities by marital duration. In any case, distinguishing age from intercourse-frequency effects should be a high priority for epidemiologists [if Hook (1985) is right and there really is a possible role for the latter], since the distinction would have practical and important implications for older women contemplating motherhood.

Royston (1982) cites one Kenyan study showing a higher prevalence of anemia in young primiparae, but the more common finding has been an association of high parity with iron-deficiency anemia. Data on singleton deliveries at 11 Indonesian teaching hospitals, for example, show an association of mother's hemoglobin in pregnancy with parity and birthweight (Table 9). Women who subsequently gave birth to infants weighing less than 2,500 grams had hemoglobin counts in pregnancy averaging 9.5, compared with 9.9 for women who gave birth to heavier infants.

Multiple Births

Dizygotous twinning is associated with maternal age and parity. Allen (1984) presents multiple birth ratios for all whites born in the United States in 1964 (Table 10). The associations with age and parity appear to be shaped like an inverted U, with peak incidence of multiple births among grand multiparae in their thirties. Why this is so is not known. There are also racial, presumably genetic, differences. Blacks in the United States in every age/parity group have a rate of twinning about 1.5 times that of whites, and rates of twinning among blacks in Africa are even higher (Allen, 1984).

Twins are more likely than singletons to be born prematurely and to be of low weight for gestational age. They also suffer higher infant mortality rates. Multiple births are not common enough to account for a very large part of the risk attributable to maternal age and high parity, but they are included here because

TABLE 9 Hemoglobin Counts in Pregnancy, by Parity and Subsequent Birthweight, Indonesia, 1978–1980

	Birthweight	
	< 2,500 grams	2,500+ grams
Parity		
1	9.8	10.1
2–4	9.4	9.9
5+	9.1	9.7
Total	9.5	9.9
	(n = 4,377)	(n = 13,747)

Source: Kessel et al. (1985).

TABLE 10 Twin Pairs per 1,000 Deliveries, by Mother's Age and Parity, United States, 1964

	Parity	
	First	7+
Mother's age		
15–19	2	—
20–24	3	—
25–29	4	9
30–34	4	11
35–39	6	11
40–44	2	5

Source: Allen (1984).

(unlike most of the intervening conditions studied here) they should be easy enough to account for in the data usually available for the study of mortality differentials in populations. (Most of the studies showing the association of biodemographic variables with infant mortality exclude multiple births.)

Complications of Delivery

The Collaborative Perinatal Study showed increasing rates of premature rupture of the membranes with maternal age: from 0.8 percent of deliveries for women aged 25 to 29 to 2.1 percent of women aged 40 to 44 (Kane, 1967). Placental abruptions also increased in frequency with maternal age and parity, when cigarette smoking was controlled for in the analysis (Naeye, 1983), perhaps due to the associations with hypertension discussed above.

Conclusions

Most of the studies that have shown excess risks of morbidity and mortality for infants born to older mothers or those at very high parity have used data from countries with low infant mortality rates, mainly in Europe and North America. In these countries increasing numbers of women are delaying childbearing into their thirties and early forties, making the consequences of older maternal age for the infant an important public health concern.

Grand multiparity, by contrast, is now exceedingly rare in these countries. In many poor countries, however, childbearing typically continues, even if it does not start, at older maternal ages, and grand multiparity is not at all uncommon. It is thus important for the design and targeting of maternal and child health and family planning programs to distinguish the effects on infant health of high parity from those of older maternal age and of maternal age as such from those of primiparity at older ages.

The mechanism for which the evidence is strongest is the effect of older maternal age on congenital abnormalities associated with chromosomal damage. As noted earlier, however, congenital abnormalities are relatively minor causes of infant death in poor countries. The same could be said of complications of labor and malpresentations, at least as direct causes of infant death. It may be that these fetal abnormalities and obstetric complications are important as contributory factors to infant deaths in poor countries, even when they do not show up as proximate causes of death. For example, for every perinatal death directly due to these causes, there may be many more infants who survive but who are weak and excessively susceptible to infectious diseases later in infancy.

The risk of pregnancy-induced hypertension rises rapidly with age for primigravidae but and less severely for multigravidae. Older primiparae are a smaller group in poor countries than in rich ones, and they include many women who have had difficulty getting pregnant or who have had prior spontaneous abortions. The extensive literature on risks to the older primiparae deals almost exclusively with low-mortality countries, although the study from central Java discussed above suggests that infants of older primiparae are clearly at high risk in poor countries as well.

The literature on "maternal depletion" in poor countries is inconclusive. (An excellent review can be found in Costello, 1986.) The best established association is between high parity and the prevalence of iron-deficiency anemia, which in turn is associated with prematurity. There is some evidence from severely malnourished communities that successively higher-parity babies are smaller at birth, but it is hard to account for this by gross maternal undernutrition. The apparent energy cost of reproduction is not great enough to account for much of a birthweight deficit. The association of high parity and low birthweight is most likely a problem of closely spaced pregnancies and is discussed further in the next section.

Most high-mortality countries do not have vital statistics reporting systems that are as complete as or that contain such detailed information as that used by Powell-Griner (1987) for the analysis discussed above. Separating the risk associated with high parity (and old maternal age) into "recurrence risk" versus "true parity and age effects" would require longitudinal data on women's reproductive careers. Such data would probably have to be gathered prospectively, since spontaneous abortions are subject to severe underreporting in retrospective self-report data, much more so than infant deaths. And prospective samples are very hard to maintain in sizes large enough to guarantee enough fetal losses and infant deaths for separate analysis by parities.

This is an important limitation of available research for the purposes of this review. The effect of family planning programs on infant mortality rates through reduced numbers of births to older women and high-parity births would presumably only operate on "unwanted" births. Women who are consciously trying to have babies in order to make up for earlier losses are unlikely to use contraception. The effect of increased contraception on perinatal death rates (and presumably neonatal death rates as well) will not be commensurate with the reduction in the proportion of births that are at high parity.

EFFECTS OF SHORT INTERVALS BETWEEN PREGNANCIES

Many studies from developing countries have found an association between the length of the preceding birth (or pregnancy) interval and the risk of infant death for the second child of the pair. Less commonly an association has been found with the risk for the first child of the pair. Mojarro and Aznar (1986), for example, found a U-shaped relationship between length of the preceding interval between births and infant mortality rates in rural Mexico, with rates declining as intervals increased and then rising again after intervals of 4 years (Table 11). Both neonatal and postneonatal infant mortality rates were higher for the shorter intervals: infants born 12 to 23 months after a live birth had neonatal mortality rates 1.6 times as high, and postneonatal mortality rates 1.5 times as high, as for infants born 24 to 35 months after a live birth. They also examined the association between short preceding birth interval and infant mortality, stratifying by age of the mother. There were very few deaths in each age/interval category for their shortest category for birth intervals (8 to 11 months), but the relative (not the absolute) risk for the 12- to 23-month intervals compared with 24+ months appeared to decline somewhat for women over 30.

It is exceptionally difficult to sort out the direction of causation and to estimate the size of effects in these relationships between pregnancy intervals and infant mortality. [Preston (1985) discusses some of the sources of confounding.] The excess mortality of those born after a short interval may merely reflect the fact

TABLE 11 Infant Mortality Rates by Preceding Birth Interval, Rural Mexico, 1965–1976

Interval Between Live Births (months)	Infant Deaths (per 1,000)
8–11	168
12–23	102
24–35	66
36–47	45
48+	56

Source: Mojarro and Aznar (1986, Table 8.4).

that this group contains a disproportionate share of premature infants, and the risk is merely due to prematurity rather than to the length of the interval. (On the other hand, as pointed out below, the short interval may itself be a cause of prematurity.) The excess mortality of those born before a short interval may be due to inadequate feeding and increased exposure to pathogens at vulnerable ages. But to estimate the importance of such effects analysts must first allow for reverse causation: the death of the first child may itself cause the subsequent interval to be short, either through a biologic effect (abrupt end of lactation leading to short postpartum amenorrhea) or a behavioral effect (grieving parents trying to quickly replace the lost child). As is true for the associations of maternal age and parity with infant mortality, the association of short interbirth intervals with infant mortality may be due in part to confounding variables, social characteristics of families that affect both the interbirth intervals and the risk of mortality independently. A likely suspect for such a confounding variable is access to health care. What has been found in rural Mexico is likely to be true of many other countries: women who use modern contraceptives tend to be the same ones who obtain good preventive and curative medical care for their children (Potter et al., 1987). Especially in the early stages of a national family planning program, the women using modern contraceptives are generally those with the most access to resources needed to promote the health of their children (Potter, 1988).[13]

Even when relationships are easy to sort out conceptually, they can be difficult to sort out empirically. Data sets from high-mortality settings, especially retrospective data, contain many errors of dating.

One example of the possible effects of nonsampling error on the estimations of the associations discussed in this paper is differential accuracy of reporting dates in retrospective data. If the birth dates of children who subsequently died are more likely to be misreported than are the birth dates of children still alive at the time of the survey, then a higher proportion of both unusually short and unusually long intervals would either begin or end with the birth of a child who later died, even if there were no true bivariate association (directly causal or due to socioeconomic confounding variables) between interval length and mortality risk. Children who died are more likely to be omitted altogether in pregnancy histories, so it is natural to expect that the reported birth dates of deceased children are more likely to be inaccurate than those of children still alive.[14] Given the magnitude and ubiquity of the association between short intervals and infant mortality from studies with various designs, it is unlikely that such reporting artifacts account for much of the observed association, however.

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[13] However, use of modern contraceptives and short interpregnancy intervals are not the same concepts. They may even be *negatively* associated. Bongaarts (1987) found in a cross-national comparison that average interbirth intervals (though not total fertility rates) tend to be lower in countries with higher rates of usage of modern contraceptives. In an analysis of micro-level retrospective data from Malaysia, DaVanzo and Starbird (1989) showed that the effects of a strong increase in contraceptive use (which would have led to longer intervals if other things had remained equal) were almost entirely negaged by the effects of a strong decline in breastfeeding. Length of breastfeeding is associated with the length of the infecund period after birth and thus, on a population level, with the length of the interpregnancy interval. Average interbirth intervals changed little in Malaysia at a time when the total fertility rates were declining steadily.

Effects on Length of Gestation and Birthweight

The association of short interbirth intervals with higher risk of infant mortality appears to be mediated, at least in part, by low birthweight and premature delivery. The evidence linking short intervals with birthweight and prematurity comes from both rich and poor countries.

It is sometimes suggested that the relationship between short interbirth intervals and high rates of infant mortality is artifactual, a result of the fact that short intervals contain a disproportionate number of premature births. Unless short intervals caused the premature deliveries, reducing the number of short intervals would do nothing to lower infant mortality rates. But this view is contradicted by evidence that short intervals are associated with low birthweight and higher risk of infant mortality even when gestational age is held constant. Pebley and Stupp (1987), for example, used prospective data from Guatemalan villages that contained information on gestational age to show that the association of short intervals from pregnancy outcome to next conception with infant mortality was similar to the association of short intervals between births with infant mortality. (Their procedure corrected, in other words, for any artifactual association by subtracting gestational age from the interbirth interval.)

Another study that defined intervals as the time between the preceding pregnancy outcome and the conception of the index child concerned a generally poor population belonging to a health maintenance organization in the East Bay area of northern California (van den Berg and Oechsli, 1984). Not controlling for maternal age, parity, or other variables, they found that 9.2 percent of conceptions less than 4 months after the previous outcome resulted in a pregnancy that terminated in less than 37 weeks. Where the interpregnancy interval was 4 to 7 months, the percentage fell to 6.8, and for intervals of 8 or more months it was 5.8 percent.

Placek (1977) analyzed data from the 1972 National Natality Survey in the United States in a way that allows separation of the association of interval length with gestational age from its association with birthweight (Tables 12 and 13). The National Natality Survey collected information for 1 in every 500 live births; only data for singletons born in hospitals to married women were presented. Table 13

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[14] Haaga (1986) presented evidence that in the Malaysia Family Life Survey the birth dates of children who later died were reported less accurately than those of children who lived.

TABLE 12 Percentage of Infants Weighing Less Than 2,500 Grams at Birth, by Preceding Birth Interval and Gestational Age, United States, 1972

Interval Between Preceding Birth and Index Birth	Gestational Age			
	< 36 weeks	37–39 weeks	40+ weeks	Total
< 12 months	43	4	3	8.8
13–24 months	27	8	3	7.5
25+ months	29	5	3	6.0

Source: Adapted from Placek (1977).

TABLE 13 Percentage of Infants by Gestational Age and Preceding Birth Interval, United States, 1972

Interval Between Preceding Birth and Index Birth	Gestational Age			
	< 36 weeks	37–39 weeks	40+ weeks	Total
< 12 months	13	38	49	100% (n = 213)
13–24 months	10	38	52	100% (n = 503)
25+ months	8	41	51	100% (n = 1,047)

Source: Adapted from Placek (1977).

shows that births after a short interval are more likely to be premature. This association could be causal, for example, if reproductive tissues have insufficient time to recuperate after one delivery. Also, a short interval could lead to cervical incompetence, which could lead to premature delivery. Table 12 shows that even within gestational-age categories infants born after a short preceding interval weigh less.[15]

In an analysis of prospective data from the Narangwal experiment in the Punjab, Fleming and Gray (1988) found an association between short preceding intervals between pregnancy outcomes and low birthweight only for boys, not for girls. The reason for a sex differential is not apparent.

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[15] Judith Fortney (personal communication) has pointed out a source of potential bias affecting studies of this association, namely, doctors' (or mothers') revision of their estimates of gestational age after the birthweight is known.

Maternal Nutrition and Fetal Growth

Maternal undernutrition is a commonly cited, almost a common-sense, explanation for the deleterious effects of a rapid succession of pregnancies. Women who live in communities where undernutrition (often seasonal) is common are known to put down much lower fat stores during pregnancy than women in rich countries or even to lose body fat during the course of a pregnancy; they also have smaller infants (NRC, 1970; Lawrence et al., 1987). Presumably it takes time after one pregnancy to rebuild stores, and the fetus of a closely following pregnancy could suffer if the mother is starting from a lower base. The energy cost of lactation is greater than that of pregnancy, so one would expect that the provision of nutrients to the first child of a closely spaced pair directly competes with the provision of nutrients to the second fetus (NRC, 1981).

Few of these causal pathways have actually been traced in human supplementation trials, and there is a good deal of disagreement in the interpretation of results from human supplementation trials and animal experiments concerning the ways in which fetal growth is constrained below genetic potential. Briend (1985) has summarized the results of supplementation trials in Guatemala, Senegal, and New York City: the energy cost of a "normal" pregnancy is about 40,000 calories, to produce a 3,300-gram baby. An additional 10,000 calories adds, on average, 30 grams to the weight of the baby (and even this is hard to document, since there are problems in the human trials of discounting all reverse causation, namely, that the women who were having bigger babies had bigger appetites). Perhaps the harshest environment for human supplementation trials to date was in rural Gambia, and there supplementation produced virtually no effect on birthweights during the dry season, but a large (approximately 220-gram) average effect for babies born during and just after the wet season. This difference was about the same as the pretrial difference in average birthweights between the two seasons. The Gambian results would have to be considered the upper limit of the size of an effect to be observed from maternal supplementation.

Some of the energy provided through supplementation is lost through (1) changes in the mother's metabolism during pregnancy (supplementation increases the energy requirement of resting metabolism during pregnancy): (2) increased body fat deposition during pregnancy (the supplemented pregnant women in Gambia gained an average of about 2 kilograms of body fat over the course of their pregnancies, compared with about 3.8 kilograms typical in rich countries and no fat gain among the unsupplemented controls); and (3) increased activity for lactating women (the husbands of the Gambian women reported increased work output by the wives who received supplementation while lactating, even though the effects on milk output were quite small). These observations suggest that the fetus and the nursing mother should be fairly efficiently protected against shortages in maternal energy intake. "It is only at very low levels [of prepregnancy weight and maternal weight gain] that the mechanisms for assuring the efficient conversion of maternal tissue into nutrients available for fetal growth are demonstrably compromised" (Kessel et al., 1985, p. 120). Winick (1983) speaks of the paradox that even undernourished women have enough nutrients in circulation to cover the apparent needs of pregnancy.

If anyone is suffering from an absolute energy shortage, it should be the mother. Yet other studies (summarized by Briend, 1985), even in West Africa, appear to show women maintaining fairly constant nonpregnant body weight through the course of multiple pregnancies. There are clinical descriptions of "maternal depletion syndrome" among poor women who have had many children, but simple anthropometry does not necessarily measure it.

Ferraz et al. (1988) analyzed data from a case-control study of singleton births in institutions in a small city in northeast Brazil. They found infants conceived after a short interpregnancy interval (6 months or less) to be at higher risk of IUGR, though not preterm delivery, than those conceived after longer intervals. The relative risk of IUGR was 1.38 for the infants born after a short interval, controlling for maternal age, education, smoking, and previous fetal loss or low birthweight. The relative risk was lowered somewhat, and lost statistical significance, when maternal prepregnancy weight was also controlled, which suggests that IUGR was caused by the maternal malnutrition associated with too short a recuperative period after the preceding pregnancy. Their ability to control for prior loss is especially helpful in distinguishing this hypothesized causal mechanism from simple recurrence risk.

Winick (1983) has suggested that the critical constraint is the ability of the placenta to transfer nutrients (rather than the availability of nutrients to the mother over the course of the pregnancy). Winick and Rosso have shown with animal studies that restricted energy intakes before and early in pregnancy lead to diminutions in the number of cells in the placenta and in cell size, which are associated with persistent neurological damage in baby rats. By contrast, starving the placenta later in pregnancy leads to smaller cell size but the same number of cells, with growth restriction but no persistent damage in the baby rats. Humans born with IUGR (but not major congenital anomalies) have placentas that weigh less than infants with appropriate weight for gestational age, due to a lower number of cells (Winick, 1967). Rosso et al. (1976) interpreted Winick's findings and the results of animal studies to mean that placental "capacity and efficiency" are affected by undernutrition at a crucial time in the first trimester of pregnancy.

A recent reviewer (Fox, 1986) doubts the evidence for gross structural problems with placentas; he and other critics of the "Winick school" adduce some evidence that the smaller fetus causes the smaller placenta rather than the other way around. Fox focuses instead on the "restricted supply of maternal oxygen and nutrients as a result of inadequate transformation of the spiral arteries into uteroplacental vessels . . . a failure of the relationship between fetal and maternal tissues at a relatively early stage of pregnancy" (p. 517).

Both lines of explanation focus attention on early pregnancy and on the maternofetal circulatory system rather than late pregnancy (when fetal growth is most rapid) and on the gross caloric intakes and the energy costs of lactation and pregnancy. For our purposes this makes more reasonable a relationship between adolescent pregnancy and rapid succession of pregnancies and fetal growth retardation. Even if it seems that there are enough nutrients to go around, placental function might be impaired if at the crucial stage of early pregnancy the mother is busy with recovery from a previous pregnancy, linear growth, and/or lactation.

Postpartum Nutrition

The postpartum nutritional status of infants born after closely spaced pregnancies would be affected if they were in competition for very limited family food supplies. Information on distribution of food intakes in families in poor countries is very hard to gather on a significant scale. A recent review (Haaga and Mason, 1987) concluded that there is fairly good evidence of discrimination against young girls in some parts of the world, especially South Asia, but there is little evidence of any tendency to discriminate against young children in general. Competition without discrimination would presumably operate to the disadvantage of both infants in a closely spaced pair. The older infant of such a pair has usually been considered the more vulnerable, since the arrival of the second infant can cause abrupt weaning. (Nutritionists always cite the example of the name *kwashiorkor* recorded by Cicely Williams in West Africa for a syndrome associated with protein deficiency, which apparently connotes the disease of the child who has been displaced.)

The problem of untangling reverse causation is difficult in demographic studies. Early weaning can itself shorten the birth interval in the absence of efficient contraception. However, Millman and Cooksey (1987) found that little of the association between short birth intervals and increased risk of infant mortality for the second infant of the pair could be accounted for by the addition of information on breastfeeding. Fleming and Gray (1988) also point out that the effects of a short succeeding interval can be spurious, confounded by the effects of a short preceding interval with which the succeeding interval is associated.

In data from the Machakos district of eastern Kenya there was a fairly constant effect (300 grams at 2 months, not statistically significant) of short preceding birth intervals on children's weight, persisting to age 36 months, but there was no consistent effect of a short succeeding interval (Boerma and van Wienen, 1984). This would tend to support the hypothesis of an effect on fetal growth rather than postpartum nutrition.

Infectious Diseases

Infectious diseases—primarily neonatal tetanus, diarrheal diseases, and respiratory infections (pneumonia and influenza)—account for the majority of infant deaths in high-mortality countries (Chen, 1983). It is not necessary, but certainly likely, that many of the excess infant deaths associated with extremes of maternal age or parity and with short birth intervals have infections as a proximate cause. There are two (not mutually exclusive) ways in which a risk factor can increase the probability of death from an infectious disease: by increasing exposure to pathogens or by lowering the resistance of the host to infection.[16] Infections could affect the fetus directly (either traveling across the placenta or, after the membranes have ruptured, through direct contact in the process of birth) or indirectly (if a maternal infection causes premature rupture of the membranes or premature delivery or retards fetal growth.) Low weight for age, both at birth and postnatally, is associated with depressed immunocompetence (Suskind and Partington, 1981) and with greater risk of death due to infectious diseases, especially respiratory infections (Kielmann et al., 1983; Barros et al., 1987a, 1987b).

How could infections differentially affect children born at different parities, or to women of different ages, or after short birth intervals? *Exposure* for the fetus would be associated with biodemographic factors: (1) if young mothers or mothers who already have a young baby (short interval) or many children in the house (high parity) are more likely to get infectious diseases during pregnancy that are known to affect the fetus or (2) if premature rupture of the membranes, more common among older women (and high-parity women?), puts a baby at greater risk of intrapartum infection. Postnatal *exposure* of the infant would be associated with biodemographic variables if (3) the presence of a young sibling (short interval) or a large number of siblings (high parity and/or short interval) increased contacts with pathogens. Postnatal *susceptibility* might be increased if (4) the infant at risk by one of the biodemographic criteria is more likely to be premature or undernourished at birth (IUGR) or subsequent to birth. The first three potential pathways are discussed below. The fourth was discussed earlier.

Maternal Infections

A fetus is better protected from infections than she or he will ever be after birth. But there are maternal infections (mostly viral) that can affect the fetus. Overall (1987) presents a useful summary of the evidence on the effects of maternal viral infections on the fetus and neonate. The four most important infectious agents are cytomegalovirus (CMV), rubella, hepatitis B, and herpes simplex. Except for rubella, which is transmitted only to the fetus, each can result in a congenital, intrapartum, or postnatal infection in the infant. CMV and herpes simplex virus have been associated with many negative outcomes for the fetus/neonate: fetal death, prematurity, IUGR, malformations, congenital infection, acute postnatal infection, and persistent postnatal infection. Rubella is associated with all these except acute postnatal infection. Hepatitis B (which is endemic in many developing countries, e.g., in Southeast Asia) also has been linked to prematurity and fetal and neonatal infectious disease (Overall, 1987).

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[16] Many infectious agents are just about uniquitous and everyone has some contact with them, but symptomatic infection (e.g., diarrheal diseases) is more likely the larger the number of pathogens that invade or the more frequent the contact with them. So "exposure" for many diseases should be thought of as "likelihood of sufficiently heavy or frequent contact."

Nonviral maternal infections are less likely to cross the placenta, but they may still affect the fetus before or during labor, especially if the membranes have ruptured prematurely.

Premature rupture of the membranes (PROM) and preterm labor in general pose a severe risk of infant mortality. Infants born after PROM are at increased risk of neonatal infections, perinatal asphyxia, and respiratory distress syndrome [though for the latter the risk associated with PROM is less than that associated with other types of premature labor (Blackmon et al., 1986)]. "Neither the etiology of premature rupture of the membranes nor of premature labor is known. However, a growing volume of experimental and clinical data strongly supports genital infection either directly or indirectly as a major cause of both" (Alger and Pupkin, 1986:760). Genital infections are more common among poor and uneducated women than among those with better access to health care and those who practice better hygiene. To the extent that the high-risk groups defined by the biodemographic variables include more poor women, they can be expected to experience premature labor more often, with negative consequences for the neonate.

Maternal parasitemias—fungal or bacterial infections that can affect the fetus—include toxoplasmosis, syphilis, tuberculosis, and trypanosomiasis.[17] In general, congenital infections may produce symptoms at birth, but in the majority of cases they are insidious, producing symptoms after some months (Berkowitz, 1984). Presumably even a congenital infection that is not itself a leading direct cause of infant deaths may render the infant more susceptible to later infection.

Ascending infections to which a baby is vulnerable after the membranes have broken include septicemia and pneumonia. During a baby's passage through the birth canal, she or he is susceptible to infections of the mother's urinary tract, including CMV and fecal bacteria (Berkowitz, 1984).

Most of these infections are prevalent in developing countries. CMV is very widespread in both rich and poor countries; one source estimates that the prevalence of CMV seropositivity in women ranges from 8 percent to 60 percent worldwide (Peckham et al., 1987). CMV has been called "a significant pathogen of the human fetus capable of producing disease ranging from subtle abnormalities not detectible at birth to severe multisystem disease" (Wright, 1980, p. 170). On the other hand, "the fate of infants born with asymptomatic CMV is not clear" (Wright, 1980, p. 170). Rubella is highly communicable and has been shown to cause severe fetal abnormalities. Urinary tract infections, very common in poor countries and among poor people in rich countries, are associated with a sharply increased likelihood of low birthweight (due to prematurity, IUGR, or both?). Sever et al. (1977) matched pregnancies in the Collaborative Perinatal Survey in the United States by maternal age, race, birth institution, and socioeconomic status and found a relative risk of 3.7 for stillbirths to women with urinary tract infections; 1.5 for a birthweight of 2,000 to 2,499 grams; 2.8 for a birthweight of 1,500 to 1,999 grams; and 4.5 for a birthweight below 1,500 grams. Gazaway and Multus (1986) reported a relative risk of preterm labor of 3.8 in pregnant women with bacterial vaginoses compared with those without it.

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[17] Malaria is discussed separately above.

What is missing for almost all of these congenital infections is strong evidence that maternal exposure or susceptibility is related to biodemographic variables. Older mothers (and grand multiparae?) are known to suffer a greater incidence of PROM, which may increase the risk of intrapartum infections. There is evidence that malaria in particular affects the growth of the fetus and the transmission of maternal immunoglobin at parity 1 (see above).

Of the other infectious agents, CMV is a good candidate for more research. There are recent studies from Britain and the United States that show the ease with which it can be passed in either direction by a nursing mother and an infant, and older siblings may bring CMV home from day care settings and infect their pregnant mothers (Peckham et al., 1987).

Increased Postnatal Exposure to Pathogens

The two most common routes for postnatal infections are fecal-oral and respiratory-respiratory (Chen, 1983; Stanfield, 1987).[18] A child born at high parity is likely to live with many siblings, and if the births have been closely spaced, these will be young siblings. A child born within a year after the previous child will reach the age at which immunities acquired across the placenta or through breast milk (3 to 9 months) when the next older sibling has become mobile and is likely to be in contact with all sorts of pathogens (15 to 21 months). Some studies of measles mortality in rural and periurban Guinea-Bissau have showed that crowding, specifically the number of other children in a household, is a better predictor of measles mortality than are anthropometric indicators of nutritional status (Aaby et al., 1983; Smedman et al., 1987). Though they do not report causes of death by birth order, the authors of the summary report on the Narangwal experiment in maternal and child health services in the Punjab report excess mortality among children above birth order seven (twice the rate for the whole sample), with most deaths in the sample caused by gastrointestinal and respiratory infections (Kielmann and associates, 1983).

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[18] The most common deadly disease transmitted otherwise than these two routes is neonatal tetanus, usually caused by septic cutting and sealing of the umbilical cord. Stanfield and Galazka (1984) summarized community surveys of causes of infant deaths, mostly from South Asia and Sub-Saharan Africa, showing that about 20 to 40 percent of neonatal deaths in these high-mortality regions are caused by tetanus. I found no literature showing any relationship between tetanus infection and the biodemographic variables of interest. Ronald Gray (personal communication) has suggested that there may be a link due to a lower likelihood of some pregnant women (e.g., young women, primiparae) receiving antenatal tetanus immunization. If so, this would represent another case in which the biodemographic variables are markers for social factors affecting maternal and child health, rather than causal factors.

Infections transmitted via feces are very important as causes of infant deaths and may be facilitated by the presence of many children, especially young ones, in the household. A recent study showed that 55 percent of cholera cases in Bangladeshi villages could be attributed to the excess risk posed by having an asymptomatic breastfeeding child who has cholera vibrio in her or his stools (Riley et al., 1987). Riley et al. surmise that careless handling of an infant's feces facilitates transmission. This would be a specific reason for young siblings, rather than just many siblings, being a risk factor, which would account for an association of close birth spacing with infant health. Similarly, another study from the International Center for Diarrheal Disease Research in Bangladesh (apparently of different villages) found infant mortality to be higher in households with more than 10 members than in smaller households—a relative risk of 1.5 in an analysis controlling for several other economic and demographic variables (Rahman et al., 1985). In this sample, the authors argue, such large households tend to have more than two adult earners and thus are not poorer than smaller households, thus reversing one effect that confounds crude household-size mortality differentials in other samples. Diarrheal diseases were the leading cause of infant morbidity and mortality in this sample, and the authors suggest that careless handling of the feces of small children explains the significant effect of household size. Yet another analysis from the same institution showed specifically that the presence of other children in the household who are less than 5 years old was associated with worse nutritional status as measured by anthropometry for boys (though apparently not for girls), even when measures of social and economic status were controlled (Becker et al., 1986). This finding is compatible with any of the three effects discussed here—prenatal nutrition, postnatal nutrition, and infectious disease.

Conclusions

Of the biodemographic risk factors discussed here, the state of knowledge about intermediate mechanisms for short interpregnancy intervals is the least satisfactory. This is especially unfortunate because the direct impact of family planning programs on infant health (apart from their association with prevention of unwanted births and better provision of maternal and child health services) is expected to come about largely from a decrease in the number of exceptionally short intervals.

In my view the best evidence is for some mechanism interfering with the development in early pregnancy of the uteroplacental circulatory system, leading to fetal growth retardation. There may also be problems with reproductive tissues (e.g., cervical incompetence, which leads to premature delivery). This latter mechanism could, at the same time, be a sort of statistical artifact (short intervals just happen to include more of the infants born, for unrelated reasons, after short gestation) and a true causal chain—inadequate recuperation from the first parturition, leaving structures too weak to support the next pregnancy. The WFS studies have found short intervals to be associated with an excess risk that continued well past early infancy, but this is consistent with both prematurity and fetal growth retardation as causal mechanisms, since infants born too small and too early who survive the neonatal period may still have respiratory and immunologic problems that weaken them later.

A high priority for further research should be the collection of information on birthweights, gestational ages, and causes of death in association with data on pregnancy intervals (preferably entire reproductive histories). This includes many variables that are extremely difficult to collect in field studies, of course, but the selectivity of hospital samples may be too great in populations where the attributable mortality risk is high enough. Where demographic surveillance systems are in place, a useful study design might be a case control, matching each short-interval death with a normal-interval death and comparing proximate causes, ages at death, and risk factors.

It would be interesting to isolate the effect on infant health of a "pure" reduction in the proportion of short interpregnancy intervals, unaccompanied by changes in women's education, their social roles, access to other health services, etc. Insofar as family planning programs actually do bring about a reduction in the proportion of short intervals, measuring this effect would allow us to estimate the benefits in terms of child health of increased effort in family planning programs. (We are leaving aside the effect of family planning programs on the number of dependents in each child's family and thus the number of competitors for presumably limited health-related resources.)

The very success of a family planning program in a new geographic area or in a poorer social class provides evidence that women's roles and their ability and willingness to use health-related resources and knowledge, among other things, have changed and are changing. To some extent the effects of confounding and unobserved variables on estimating the effect of interval length on infant mortality can be alleviated by better measurement of social, economic, and other health-related variables and by careful specification of statistical models. Such research will help policymakers assess the most effective mix of components of health services (including family planning) in different settings.

ACKNOWLEDGMENT

I received very helpful advice, comments, and references from many members of the working group and from others who reviewed earlier drafts of this paper. In particular, I would like to thank Julie DaVanzo, Peter Donaldson, Judith Fortney, Ronald Gray, Jorge Martinez-Manatou, Anne Pebley, Joseph Potter, and James Trussell for all of their assistance.

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Psychosocial Consequences to Women of Contraceptive Use and Controlled Fertility

Ruth Dixon-Mueller

Recent research on the consequences of contraceptive use and controlled fertility has focused almost entirely on the physical health of women and children as measured by rates of maternal and child morbidity and mortality. Little attention has been paid to the broader implications of trends and variations in the reproductive behavior of women—that is, to their psychosocial consequences—as reflected in the way women in differing socioeconomic circumstances feel and talk about themselves and their family and work situations. Given the evidence on the connections between psychosocial stress and physical disorders (albeit mostly from industrialized countries), it is remarkable that the literature on health consequences of contraceptive use and controlled fertility essentially ignores this component of health and well-being. Indeed, neither the multicountry World Fertility Survey nor the Demographic and Health Survey includes questions that would permit analysis of the relationship between reproductive behavior and psychosocial stress or disorder.

In examining the health consequences for women of contraceptive use and controlled fertility, we should adopt the broadest conceptual approach in order to capture the full range of costs and benefits of different reproductive behaviors. Women alone bear the physical risks of pregnancy and childbirth. Women also bear the physical risks of using female methods of contraception—the oral pill, the intrauterine device, injectables, and tubal ligation, for example—and of vol-

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untary pregnancy termination as well. But the calculus of health benefits and costs cannot stop here. For many women the psychosocial aspects are at least as salient as the purely physical aspects, if not more so, in both their positive and negative manifestations. Of particular importance are levels of psychosocial stress associated with women's concerns about the timing or spacing of their births, about subfecundity or excess fertility, and about the use of specific methods for avoiding unwanted pregnancies.

Concepts of health and illness are highly culture bound, of course. Developing a set of indicators of how respondents perceive their physical health, yet alone their mental health, would be extremely difficult. Nevertheless, such conceptual and methodological difficulties should not prevent us from incorporating this crucial aspect of women's reproductive lives into our theoretical frameworks and, subsequently, into our data collection as well. Doing so would help to narrow the gap between current research on morbidity and mortality and the World Health Organization's definition of health as a "state of complete physical, mental and social well being and not merely the absence of disease or infirmity."

The purpose of this paper is to suggest a framework for thinking about the psychological consequences to women of contraceptive use and controlled fertility. The literature based on empirical studies is scant. Instead, this paper draws primarily on common-sense possibilities and on ethnographic reports of how women talk about their lives.

CONCEPTS OF HEALTH AND WELL-BEING: INDICATORS OF PSYCHOSOCIAL STRESS

Stress, whether chronic or episodic, may be categorized as *systemic* (i.e., primarily physiological); as *psychological* (i.e., primarily cognitive); or as *social* (i.e., induced by the disruption of some social unit or system).[1] For convenience the concepts of psychological and social stress are combined in this paper. Psychosocial stress can manifest itself in at least three types of mental disorder: ***depression***, ***anxiety***, and ***hostility***, which, in some Western populations, at least, constitute distinct dimensions, or subscales, of mood. Each type of disorder is relevant to the analysis of health consequences to women of contraceptive use and controlled fertility. Each may also be related to—or perceived and expressed as—physical disorders, as, for example, when a woman anxious about whether she can provide adequate food for her children complains of dizziness, headaches, or nervous exhaustion.

Depression is usually assessed by respondents' self-reported emotional symptoms, such as feelings of despair, helplessness, worthlessness, shame, being in a

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[1] This discussion draws generally on the literature on the connections between psychosocial stress and physical disorders, notably Levine and Scotch (1970), Monat and Lazarus (1977), Cohen and Syme (1985), and Barnett et al. (1987).

"low" mood, etc., and by physical symptoms such as lethargy, marked changes in eating or sleeping habits, and other indicators. Anxiety is measured by feelings of generalized or specific worry, fear, and intense dread or foreboding as well as by physiological indicators such as raised blood pressure or increased heart rate. *Hostility* is represented by feelings of anger, conflict, or hatred, as expressed perhaps in violent, erratic, or abusive behavior toward self or others or in social withdrawal, with associated physical symptoms.

The discussion in this paper will be directed primarily to the phenomenon of *anxiety*, which, cross-culturally, is probably the most common disorder that women experience in relation to their sexual and reproductive capacities. As one reviewer (Harrison, 1983; see also Ford, 1964) of the cross-cultural literature on pregnancy and childbirth concludes:

> The major, single, unifying theme that runs through both cultural accord and divergence on the subject of childbearing is that the entire experience is not only a time of discomfort but a time of danger, the source of which may be physical or supernatural. In consequence, it is a time of vulnerability and anxiety, and the penalties for failure are high (p. 69).

The major focus of this anxiety, I propose, is the way in which threatening reproductive events or conditions, such as failure to bear a child, fear of an unwanted pregnancy, or expectation of ill health from contraceptive use, affect women's perceptions of their ability to perform those essential social roles upon which their survival, security, and well-being depend. The penalties for failure are indeed high, and they are firmly embedded in the social system.

PSYCHOSOCIAL STRESS AND ROLE PERFORMANCE

The relationship between social structures, social roles, gender, and psychological distress has been studied in Western populations (Barnett et al., 1987). But what approach is feasible for comparative analysis? Studying a sample of educated Ghanaian women, Oppong and Abu have proposed a framework that is useful for analyzing the impact of contraceptive use and controlled fertility on women's lives (Oppong and Abu, 1985; see also Oppong and Abu, 1987).[2] Their approach identifies seven distinct social roles that affect—and are affected by—reproductive behavior: the maternal, occupational, conjugal, domestic, kin, community, and individual roles. Elaborated further below, each of these roles involves particular sets of activities, expectations (rights and obligations), and social relationships; different patterns of decision making; the investment of time and other resources; and the possibility of psychosocial and perhaps economic rewards. These various attributes can best be described as *role content.*

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[2] For related approaches to role performance and stress based on U.S. studies, see Aneshensel and Pearlin (1987) and Barnett and Baruch (1987).

In addition, the seven roles in combination represent a *role profile*. Like role contents, role profiles include normative elements that are common to the social group (however the group may be defined) (see, e.g., Mason, 1983) and elements that are unique to each woman. Ascertained through individual interviews or focus group discussions, a role profile represents in configuration a woman's perception of the varied demands made on her and what aspects of her life she finds most difficult and most satisfying. The profile allows a researcher to identify which roles have the highest priority at any given time in a woman's life cycle, which roles offer the least and the greatest rewards, and which roles constitute sources of role strain and role conflict.

The concepts of role strain and role conflict are particularly important as potential sources of psychosocial stress that may result in anxiety, depression, or hostility. Role strain refers to the extent to which a woman feels unable to cope with the demands of a particular role with the resources at her disposal (e.g., time, energy, money, or social networks) (Oppong and Abu, 1987). Role conflict refers to the extent to which a woman perceives the demands of two or more roles that she occupies simultaneously as incompatible (e.g., working outside the home and caring for young children). In a slightly different formulation, role conflict can also refer to the extent to which people disagree among themselves about the normative content of a particular role, for example, when a mother-in-law and husband (or a husband and wife) hold contradictory views of what it means to be a "good wife."[3] The former definition is more salient to this paper.

The seven-roles framework presents a useful analytic approach for investigating the psychosocial consequences to women of contraceptive use and controlled fertility. In every society reproductive events or conditions such as menstruation, sexual intercourse, the use or nonuse of a contraceptive method, the birth of a child, breastfeeding, or the inability to carry a pregnancy to term are usually highly charged with personal and social meaning. Indeed, reproductive events often form the object of elaborate social ritual (Paige and Paige, 1981). Whether their net impact on women is positive, negative, or neutral depends in large part on how such events or conditions transform the content of particular roles (as women perceive them) and the nature of the overall role profile; that is, it depends on the resulting waves or ripples that flow through the "grid" of a woman's multidimensional role performance (Figure 1). It follows that reproductive behaviors that *intensify* rather than reduce role strain or role conflict—particularly among those roles that a woman defines as most salient to her security and survival—are likely to be perceived as the most stressful.

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[3] This definition of role conflict is not drawn from Oppong and Abu's work.

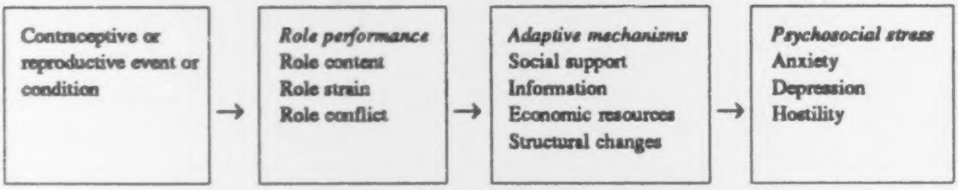
Contraceptive or reproductive event or condition
Role performance
Role content
Role strain
Role conflict
Adaptive mechanisms
Social support
Information
Economic resources
Structural changes
Psychosocial stress
Anxiety
Depression
Hostility

FIGURE 1 Model of the influence of contraceptive and reproductive patterns on stress.

Stress may be mediated by adaptive, or "coping," behavior, particularly in settings where women can draw on strong social support networks or on other material and social resources to counter or dilute the negative effects of threatening events or conditions (Cohen and Syme, 1985). Intervening mechanisms include relying on interpersonal networks (e.g., seeking social support for a decision to terminate a pregnancy); having access to economic resources (e.g., being able to give adequate food and shelter to a newborn child); receiving accurate information (such as learning about the probability of contraceptive side effects); and controlling outcomes by making structural changes (such as finding a good child care provider). Despite such adaptive strategies, however, it is probably safe to say that most women in most socioeconomic circumstances are likely to experience extreme stress at some time in their lives that is triggered by sexual or reproductive events or conditions. The resulting psychosocial distress may or may not translate directly into physical disorders. I suggest, however, that its effects are likely to be no less pervasive than those mortality and morbidity indicators with which we are most familiar.[4] When we consider that poverty, powerlessness, and physical illness are also identified as major sources of psychosocial stress, we can better understand how certain reproductive events—interwoven as they are with other insecurities and threats—can engender such high levels of emotional disturbance, particularly among low-income women.

CONTRACEPTIVE AND REPRODUCTIVE PATTERNS AS POTENTIAL STRESSORS

When we shift our attention from the effects of contraceptive use and controlled fertility on women's physical health to women's psychosocial or emotional well-being, we must also include those factors affecting child health and survival because these become matters of critical concern for women. The "wantedness" status of a particular pregnancy or birth is particularly relevant here, for obvious reasons. The fear of miscarriage, stillbirth, birth deformity, or infant death or illness is a major cause of anxiety among women with wanted pregnancies, while an unwanted pregnancy or birth can drive women to attempt a dangerous self-induced abortion; to infanticide; or to prolonged hostility, depression, and despair. Similarly, we must include under women's health not only reported rates of maternal morbidity and mortality associated with contraceptive use and childbirth but also—and perhaps more importantly—women's *fears* of such conditions or events: the fear of getting pregnant accidentally, for example, or of not getting pregnant at all; the fear of potentially debilitating contraceptive side effects; and the fear of trauma or death in abortion or childbirth. As noted above, a woman is most likely to focus such fears and anxieties on her ability to perform those social roles described below that are most salient to her immediate concerns with security, survival, and general well-being.

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[4] Harrison (1983) presents a fascinating study of the pervasiveness of psychosocial stress related to (in decreasing order of frequency) high fertility, contraception, and fetal wastage among a sample of women living in poverty in rural El Salvador.

Conjugal Roles

Conjugal roles define a woman's relationship with her sexual partner(s), whether in a casual or "visiting" union, a consensual union (cohabitation), or a formal marriage (monogamous or polygamous). Oppong and Abu (1987) found that educated married women in their Ghanaian sample ranked their level of satisfaction with their conjugal roles on average below their parental, occupation, kin, and individual roles. They also ranked their level of "role deprivation"—that is, the gap between what they expected of their conjugal roles and what they experience—as significantly higher than for any other role. Yet women defined the conjugal role as high priority (second only to the parental role) for their social standing and personal happiness.

Patterns of contraceptive use and controlled fertility should have an immediate impact on the conjugal relationship. Some research has attempted to identify qualities of conjugal roles that influence sexual and contraceptive practices and fertility outcomes (e.g., the effects of joint vs. segregated role relationships, male-dominant patterns of decision making, female autonomy) (see, e.g., Rainwater, 1960, 1965; Fawcett, 1973; Luker, 1975; Miller, 1986). Little systematic research has been done that turns the causality in the other direction. For example, how do different patterns of contraceptive use (including indigenous methods or no method at all) affect a woman's conjugal role performance and the levels of stress associated with her relationship with her sexual partner?

Ethnographic literature from both industrialized and developing countries suggests that the conjugal relationship itself is frequently a source of psychosocial stress.[5] Houston's (1979) informal interviews with rural and urban women, both literate and illiterate, in six developing countries sometimes elicited ambivalent if not negative statements from the women about men in general and their own marriages in particular.[6] It is thus not surprising that contraceptive behavior can also be difficult, emotional, and conflicted for a number of reasons. Some conflicts relate to the question of who has the *right* to use—or who should assume the *responsibility* for—birth control; others relate to manifest or latent disagreements about sexual practices, childbearing intentions, or other aspects of the conjugal relationship such as women's resentment of male power and privilege or of their husband's drinking, philandering, or failure to support the family. For a woman who wants to delay or terminate childbearing, the unwillingness of her partner to use birth control consistently or at all can cause fights and strong feelings of anxiety, depression, or hostility. Wives who derived little sexual satisfaction from their husbands were particularly likely to be resentful about birth control in a study of lower-class families in the United States (Rainwater, 1960). The powerlessness of a woman in Sri Lanka was expressed this way:

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[5] For a review of the literature on the family in industrialized societies, see, for example Croog (1970).

> What is the good of refusing [a husband's sexual demands], they will never let us alone. [If I refuse] he will go to some other woman and then what will become of me and my children? (Ryan, 1952:376)

Or, in a resentful mood, a Kenyan woman commented:

> The men could not care less about family planning—you never see a man going to a family planning clinic. Some, I think, would like to have more children to keep the women at home (Huston, 1979:144).

Women who were focus group participants in a study of attitudes toward natural family planning in the Philippines complained that it was difficult to get their husbands to cooperate in periodic sexual abstinence when the man got drunk and became sexually aggressive or lost control (Verzosa et al., 1984). A woman's fear of unwanted pregnancy naturally extends to a fear of intercourse, but refusing sex poses its own threats and anxieties. Among a sample of women attending a family planning clinic in Lebanon, well over half said they had refused on some occasions to have intercourse during the previous 5 years because of fear of pregnancy (Chamie, 1977). The anger of some Latin American women who complain of their menfolk—"he uses me"—often centers on men's refusal to practice birth control despite persistent sexual demands. One study of attitudes toward family planning in Mexico City found in focus group sessions that a major cause of women's resentment about their marital situation was a "sense of deep depersonalization, humiliation, and physical dissatisfaction" caused by their husbands' treatment of them during sexual relations (Lyon et al., 1981).

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[6] Huston interviewed rural and urban women, literate and illiterate, in Egypt, Kenya, Sudan, Tunisia, Sri Lanka, and Mexico. For similar comments on the contradictions and instability of conjugal roles as women perceive them, see Harrison (1983) and Beneria and Roldan (1987).

In some circumstances women's negative attitudes toward sexuality and toward their partners could be moderated when the fear of unwanted pregnancy is removed. Indeed, this should be one of the major psychosocial benefits of cooperative and effective contraceptive use, depending on the method used. We have little evidence on the effects of different methods on women's sexual enjoyment and orgasmic capacity, but there is some suggestion that the use of coitus-dependent methods such as withdrawal or the condom may reduce the frequency of intercourse and/or the sexual pleasure of one or both partners as compared with coitus-independent methods (see, e.g., Verzosa et al., 1984; Coleman, 1981). In any case, women whose husbands can be trusted to practice birth control consistently often express more positive attitudes about the conjugal relationship. The expression "he takes care of me" compared with "he uses me" suggests far more than relief of anxiety about bearing an unwanted child.

A woman who decides on her own to use birth control—either in accordance with or in defiance of her partner's wishes—incurs a different type of stress from the woman dependent on her partner's cooperation. On the positive side, she may feel in control of her body and confident about her ability to determine her own sexual and reproductive behavior. But depending on the method used and on what she has heard or experienced, her anxieties about real or rumored side effects and about accidental pregnancy can adversely affect her own well-being and the conjugal relationship. Moreover, she may be resentful at having to carry the burden of health risks in order to be sexually available to her partner (especially if she experiences little sexual pleasure herself), or she may be anxious about other effects of contraceptive use on the relationship, such as her partner's perceptions of her as being "like a prostitute" or "no longer a woman" (Warwick, 1982, p. 113). If she uses a method against her partner's will, she may be accused of sexual promiscuity or be threatened with violence, desertion, or divorce. The extent to which such threats become personally devastating depends on her access to adaptive mechanisms such as economic resources and social support.

The effects of controlled fertility on conjugal roles are also pervasive. Postponing the first birth can eliminate the psychosocial stress associated with a premarital pregnancy that results in an out-of-wedlock birth or a forced marriage in which one or both partners feel trapped or the stress incurred within marriage by an early birth for which a couple is emotionally or financially unprepared. Similarly, child spacing and limitation may place less stress on the relationship between sexual partners, other things being equal, leaving more time for couple-oriented activities. This should result in less role strain or conflict for women and in higher reported levels of satisfaction with their conjugal relationships.

In settings where family elders place a high value on a first birth soon after marriage and on more frequent childbearing, however, a couple's (or woman's) decision to postpone or limit births may produce some anxiety in the conjugal role as it conflicts with the kin role. Being a "good" wife in this sense conflicts with being a "good" daughter, daughter-in-law, or family member. Moreover, in circumstances where women are anxious that their husbands might leave them, frequent childbearing may be seen as a way to prevent desertion or divorce. In this sense, then, controlled fertility could leave women feeling more vulnerable. Such fears relate not only to the number of children born but also to child survival. As an Egyptian woman in a village in the Nile Delta said to an interviewer,

> Of course it's important to have more than one child. Do you know what my husband did after our first two children died, one after the another? He went to his mother and asked her to find him another wife (Warwick, 1982:109).

Less drastically, respondents in the multicountry Value of Children surveys sometimes stressed the importance of children for strengthening the marital bond (Fawcett, 1983). It is not clear how this might be linked to the timing or number of children, however.

Occupational Roles

Perhaps the most clear-cut area in which contraceptive use and controlled fertility can reduce psychosocial stress is in women's educational and occupational roles. Anxiety about having to interrupt or terminate schooling or employment because of an unwanted pregnancy can certainly be alleviated by the practice of safe and effective contraception and the availability of safe abortion. Effective contraception introduces an element of choice, or control, that is generally associated with lower levels of stress. In particular, contraception or abortion that results in the postponement of a first birth reduces the likelihood of a woman having to drop out of school prematurely and either stay home to care for the child or seek low-wage employment in order to provide minimal economic support. A study of adolescents in Ibadan, Nigeria, found that almost half of never-married female secondary school and university students had become pregnant. Of these, virtually all of the university students and approximately 80 percent of secondary school students chose to terminate their first pregnancy by induced abortion so that they could stay in school (Nichols et al., 1986).

Similarly, postponement of a first birth and the spacing and limitation of subsequent births (or the avoidance of childbearing altogether) frees women to pursue employment less encumbered by the stress of conflicting child care demands and with more time and energy to invest in occupational role performance, other things being equal. It also helps to avoid interruptions in employment that can harm the future prospects of not only the minority of women in "career" positions but also, in some cases, women in wage work if jobs are hard to find

(Standing, 1983). A study of low-income mothers of young children who participated in the Stress and Families project in Boston, Massachusetts, reported that "the most common reasons given for stopping work were events such as pregnancy, birth of a child, or problems with child care arrangements" (Belle and Tebbets, 1982, p. 184). In general, the women associated work with "confidence, self-esteem, accomplishment, dignity, and independence." Women who wanted to be working but who were unable to do so experienced more symptoms of depression than did employed women or those who did not want to work (Belle and Tebbets, 1982).

Whether fertility limitation is actually translated into more intensive or extensive schooling and employment and into greater self-esteem deriving from these roles depends, of course, on the priority a woman places on the occupational role relative to other roles, on the structure of the labor market that determines the demand for her labor and the returns she is likely to earn, and on her class position, among other factors. The women interviewed by Huston in six developing countries perceived themselves as "having primary responsibility for the economic wellbeing of their families . . . provided that society gives them the opportunity to participate in income-generating economic activities" (Huston, 1979, p. 147). Indeed, they defined the opportunity to work for pay as of top priority in solving their economic problems, followed by education that would lead to employment, and access to family planning (Huston, 1979). In turn, whether fertility limitation actually reduces the stress of combining employment with childbearing depends on such factors as the time and locational flexibility of the job and the availability and cost of acceptable child care.

Several qualifications to the generalization about controlled fertility reducing psychosocial stress associated with occupational roles are in order here. First, in certain circumstances having fewer children or having children of the "wrong" sex may actually interfere with the performance of a woman's occupation, thus creating role strain. Examples include secluded Hausa women in northern Nigeria who depend on their children to sell foodstuffs that the women prepare in their homes or West African women traders, beer brewers, oil pressers, or independent cultivators for whom children often provide essential labor. Occupational stress induced by a shortage of child labor can often be resolved, however, by structural changes such as employing other family members, hiring wage workers, or fostering the children of kinswomen.

In addition, as Standing (1983) emphasizes in his review of the relationship between women's employment and fertility, in some circumstances "childbearing and childraising may in fact not constrain the labor force participation of women, and may have negligible opportunity costs" (p. 519). The general argument here is that some occupations, such as home-based manufacturing (crafts) or agricultural work, are compatible with child care. Conclusions such as these are based on analyses of the relationship between employment and reproductive patterns, however. Missing is an analysis of the stress that can be engendered by attempting to breastfeed an infant or keep track of toddlers while engaging in productive activities that must constantly be interrupted.

Maternal Roles

Perhaps no role presents women with such rewards and anxieties as motherhood. A sample of educated Ghanaian women ranked the maternal role as of highest priority for their well-being (an average score of 2.9 of 3.0) and as most satisfying (2.5 of 3.0); at the same time motherhood was associated with high levels of strain (inadequate resources of time and energy, among others) and with a considerable gap between expectations and reality (Oppong and Abu, 1987). Any parent will quickly identify with the stress as well as the pleasures of rearing children. As a woman interviewed in Mexico remarked, "My children are my greatest source of happiness, but also my greatest worry" (Warwick, 1982, p. 113). The theme of women's ambivalence about the maternal role appears throughout much of the ethnographic literature.[7] Cross-cultural demographic research on the value of children suggests that wives often place more importance than husbands do on the social and economic benefits from children but are also more sensitive to the personal costs of raising them (Fawcett, 1983).

The question of interest here is whether contraceptive use and controlled fertility might contribute to lower levels of psychosocial stress (anxiety, depression, hostility) associated with pregnancy, childbirth, and parenting. This paper does not address the issue of involuntary subfecundity or sterility which can produce extremely high stress levels among individuals or couples desiring to become biological parents.

The psychosocial benefits of contraceptive use and controlled fertility to women who are at high risk of pregnancy-related disabilities or even death are obviously great. These risks may be associated with a woman's individual characteristics, her class position, or the environment of health care services. Women often express fear of disability during pregnancy and of excruciating pain or death in delivery.[8] Women also express anxieties about miscarriage, stillbirth,

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[7] For a review of some of this literature and an illustration from El Salvador, see Harrison (1983, pp. 62-75).

[8] As Harrison (1983) notes,

> Childbirth is often prolonged and painful, particularly for primiparas, and all societies have developed special techniques for dealing with difficult births. . . . Fear of pain is accompanied by apprehension about wholeness and viability of the fetus, about whether fetal presentation will be favorable to an easier birthing, and about survival itself. The ethnographic evidence simply does not substantiate the claim that the fear and pain associated with childbirth is an artifact of Western civilization (p. 69).

birth defects, and infant and child death, all of which have the potential of causing intense emotional distress. Other things being equal, controlled fertility should contribute to reductions in psychosocial stress associated with entry into the maternal role.[9]

The ethnographic literature also suggests that women are often extremely worried about their ability to provide adequate care for their children, are depressed at their apparent inability to do so, or are hostile about the failure of other persons or institutions to help them. At the minimum, adequate care means sufficient breast milk and food, clothing, shelter, and protection from accidents, illness, and death. Beyond sheer survival it means economic resources for medical care, schooling, and other investments; time and energy for individual attention to each child; appropriate socialization to age-related responsibilities; and the transmission of cultural values and practices.

In general, one would assume that voluntary postponement of the first birth, longer spacing of subsequent births, and the termination of childbearing at an earlier age would ease the stress of the maternal role by reducing women's anxieties about providing adequate care. In Huston's (1979) interviews in six developing countries, for example, most women considered smaller families (however defined) to be more desirable and identified the availability of modern contraceptives as a positive change compared with their mothers' lives:

> In expressing their views about planning their families, the women seemed to be less concerned about themselves than about their ability to provide for their children [especially education for good jobs]. When they did speak about themselves, it was in terms of their health and the fact that child spacing would give them strength (p. 135).

On the other hand, controlled fertility may not reduce the stress associated with raising children if role expectations escalate with regard to the time or intensity of maternal investment along with aspirations for children's "success." The oft-cited anxieties of Japanese mothers who are preoccupied with their children's academic achievements starting even prior to their enrollment in kindergarten is just one example. In other words, the nature and severity of the stresses relating to maternal role performance are strongly related to social class and cultural expectations. In addition, as Figure 1 suggests, stresses induced by maternal role strain or role conflict can be mediated by adaptive mechanisms such as social support networks (e.g., assistance from a husband or other family members with child care) or structural changes.

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[9] Recent research suggests that the effects of later first births and lower parity on maternal and infant deaths in some developing countries may be small, however. See, for example, Winikoff and Sullivan (1987) and Trussell and Pebley (1984).

Domestic, Kinship, and Community Roles

Space does not permit a detailed analysis of the possible effects of contraceptive use and controlled fertility on each of these roles. A few points will be highlighted here.

The domestic role refers to women's housekeeping obligations such as cooking, cleaning, washing, shopping, etc. The sample of educated Ghanaian women ranked this sixth of the seven roles in priority, the lowest in derived satisfaction, and the highest in role strain (inadequate time, energy, and money) (Oppong and Abu, 1987). Women throughout the world complain particularly of the stresses of the "double day," when conjugal, maternal, and domestic role expectations are heaped on top of occupational roles with little modification. Women's anger is often palpable:

> I work in the field, opposite the men, seven hours of hard work, and then I go home, and I am required to play the role of housewife 100 percent, cleaning, and washing for the children (Huston, 1979:135).

> I am working outside and inside. I am doing a dual job. Some people think that work is liberation of women. It is not liberation. Sometimes it is just that women are more exploited (Huston, 1979:135).

The extent to which controlled fertility eases the stress of this role depends on many factors, including household size; age and sex composition; standards of upkeep; and the availability of additional help from household members or from other relatives, neighbors, or paid workers. In general, one would assume that having fewer children and spacing them further apart eases domestic role performance. Yet, as in the case of child care, standards for domestic role performance can escalate with rising incomes, among other factors. Moreover, the literature on sex preferences and the value of children indicates that most women hope to have at least one daughter even in countries with very strong preferences for boys, primarily (but not solely) because girls help around the house more than boys do. In this sense, then, the failure to bear a daughter could exacerbate domestic role strain, especially if there are many sons and little help from others.

The kinship role refers to a woman's relationship with her affinal and consanguineal kin and in some cases with "fictive kin" (e.g., godparents to herself or her children) upon which much of her social and economic security and survival may depend. Good relations with kin can endow women with considerable satisfaction and self-esteem. On the other hand, the kin role can be highly conflicted. A woman may resent her husband's filial obligations to his parents or other elders if they conflict with his conjugal obligations to herself and their children, for example. She may be caught in a dispute between her own and her husband's kin over a dowry or bride price, with her husband's brothers over property when she becomes widowed, or with cowives over the allocation of resources to children in a polygamous marriage. The expectations of kin regarding a woman's contraceptive practices and childbearing may conflict sharply with her own wishes, particularly when elders expect to maintain control over the sexual and reproductive decisions of the younger generation through mechanisms such as arranged marriages and extended household residence.

In speculating on the potential impact of contraceptive use and controlled fertility on kin relations, then, we would have to know whether such behavior is generally supported by significant kin, ignored, or specifically opposed and criticized. A woman whose behavior is viewed as deviant may experience considerable guilt, anxiety, or hostility when she seeks contraceptive services or "fails" to produce a child, or the socially "appropriate" number of children, or a son. Such examples are common. A wife interviewed in the Nile Delta region in Egypt commented:

> The bride must get pregnant right away. I stayed four months after marriage without getting pregnant. I was very worried. Everyone was anxious to find out whether I was pregnant or not. Every time I had my period my husband's family talked about me (Warwick, 1982:109).

On the other hand, fulfilling the reproductive expectations of the kin group often brings women real satisfaction:

> My husband's family treated me differently after I had the first child. I felt settled and secure among them. I became one of them. They all called me "mother of Hassan" (Warwick, 1982:109).

We might include women's concerns about who will support and take care of them in their old age under the general topic of kin roles, although such concerns relate more specifically to the maternal and conjugal roles. The absence of sons or daughters who are able and willing to care for an aged parent creates a strong potential for anxiety or depression among women without alternative means of support. In the Egyptian study noted above, "the fear of being a widow without children, and especially without sons [was] one of the strongest motives for women to produce more children than they might ideally desire. [Without sons, a woman] does not feel at ease (Warwick, 1982:113)." The absence of children for support in old age is likely to strain women's relations with consanguineal or affinal kin as well, for one of those relatives may have to take on the responsibility of her care, willingly or not.

Beyond the kin group, women play a role in community relations that may be broad or limited and of high or low priority, depending on many factors. As noted in the discussion of kin roles, contraceptive use may or may not be stressful depending in part on community attitudes and practices: the judgment of religious leaders, for example (and the salience of these to the individual or couple), and the prevalence of contraceptive information and use in the community.

Community activities include participation in religious and ceremonial events, political meetings, sports and recreation, entertainment, community projects, and general public socializing, among other events. Fertility limitation may or may not facilitate women's involvement in community activities. In some respects it could act in a manner similar to women's occupational roles. In other respects, however, having more rather than fewer children could encourage community involvement, given that many community activities are child related and children often involve adults in social interactions that transcend other roles. These community roles could be stress inducing if they conflict with other roles or they could be stress reducing if they integrate women into supportive social networks, increase women's access to other resources, and heighten women's sense of community identity and self-esteem.

Women as Individuals

The role of "individual" is listed last, not because it is unimportant but because for many women, especially those with large families living in poverty, it is the role that women have the least time for. The content of this role includes activities such as pursuing personal interests; relationships with friends; and identities relating to women as persons, as individuals, as "themselves." The educated Ghanaian women rated the role as lower priority on average than their parental, conjugal, occupational, and kin roles (in that order) but among the most satisfying (Oppong and Abu, 1987).

It is difficult conceptually to separate a woman's individual role from others because her self-perception often derives primarily from her roles as wife or lover, worker, mother, daughter or daughter-in-law, and so on. Nevertheless, several aspects of the individual role deserve mention here.

The first relates to how contraceptive use affects women's self-perception. As mentioned previously, it can contribute to heightened self-esteem where women believe they are now in greater control over their sexuality and reproductive capacity and thus, perhaps, over other aspects of their lives as well. Yet contraceptive use also has psychosocial costs to women as individuals.[10] One involves fears about health consequences of contraception, abortion, or sterilization, based either on rumor or fact. Depending on the method, these fears include but are not limited to dizziness, physical weakness, pain, nausea, altered menstrual flow, cancer, bodily damage, temporary or permanent sterility, and even death. They also include concerns about whether to continue or discontinue a particular method. A second cost relates to worries about using a method correctly or consistently, such as taking a pill every day or inserting a diaphragm or cervical cap correctly, and the associated self-perception as a person who is "unnaturally" or perhaps "sinfully" trying to avoid or terminate a pregnancy. A third cost relates to the psychosocial stress of obtaining services and supplies.[11] Encounters with service providers often cause great emotional distress, especially in clinic settings requiring pelvic examinations where clients feel frightened, confused, humiliated, and shamed. Women attending urban clinics in Morocco, for example, complained bitterly of degrading and dehumanizing treatment by clinic personnel:

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[10] The following points are discussed in greater detail in, among many others, Scrimshaw (1976), Hollerbach (1982), and Bruce (1987).

> If you make a mistake, if you mispronounce a word, the name of a syrup or a pill, the nurse laughs at you, calls her colleagues to tell them the story and points at you. You feel the floor crumble away under your feet (Mernissi, 1975:424).

Another woman adds:

> When we are waiting to get into a gynecological service, they will shout at us. "Take off your pants before going into the doctor's office." You take off your pants in the hall and sit there waiting. There are . . . people walking by. You feel inhuman (Mernissi, 1975:424).

Clearly, then, psychosocial stress induced by the act of obtaining birth control services should clearly be considered in any cost-benefit analysis in addition to the stress engendered by their use.

Finally, there is the question of how women's roles as individuals are affected by fertility limitation. Delaying the first birth, spacing subsequent births, and limiting the total number should leave women more time to pursue their own interests as individuals (other things being equal) and perhaps to develop a stronger individual identity. This is particularly true of women who avoid childbearing altogether. Yet whether fertility limitation translates into a more individualized role or a more positive or negative self-image for women depends on many factors.

Much depends on the structural and cultural opportunities provided to women for individual role behaviors in each society and on the relative priorities that a woman herself places on the individual role compared to others (particularly, marriage and motherhood) in her role profile. Where opportunities are plentiful and priorities high, fertility limitation should reduce individual role strain and thus the stress engendered by having "no time for myself" or wondering "who am I?"

Yet marriage and motherhood are highly valued in all societies. Women who remain childless by choice or who have one or two children may be viewed as selfish, irresponsible, or unnatural, while those with larger families are often seen as warm, loving persons who are willing to make sacrifices and take on adult responsibilities.[12] (Having "too many" children may also be viewed as selfish or irresponsible in some settings, however.) Women may consequently experience considerable anxiety about their worth as persons if their reproductive behavior deviates significantly from the social norms of their group. Once again, however, psychosocial stress can be diluted by the adaptive mechanisms identified in Figure 1. In particular, if the environment offers some choice and a woman has the resources, she can seek out those activities and social networks that offer the strongest confirmation of her role as an individual.

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[11] For a review of literature on provider-client transactions, see Lapham and Simmons (1987).

CONCLUSIONS

With few exceptions the demographic analysis of the health consequences to women and children of contraceptive use and controlled fertility has essentially ignored the question of mental health.[13] Yet the psychosocial consequences to women of contraceptive use and controlled fertility—both positive and negative—are no less compelling than the consequences to their physical health and life chances.

Because most reproductive events and conditions are highly charged emotionally and socially, it is essential that the psychosocial as well as physical consequences of reproductive behaviors be routinely included in any calculus of health benefits and risks. In a parallel fashion it is not just maternal mortality or morbidity that are relevant but also the broader notion of reproductive mortality or morbidity (or, conversely, reproductive health), which encompasses the health risks and benefits involved in attempts to prevent pregnancy as well as of pregnancy, childbirth, and sexual behaviors.[14] Third, the question of how women and men throughout the population perceive and interpret health consequences may be as important to our understanding as are the formal measures of morbidity and mortality routinely collected.

Three distinct (although interrelated) dimensions of psychosocial stress have been identified in this paper: anxiety, depression, and hostility. Clearly, such attitudes and behaviors are not easy to measure. Some women may express such feelings verbally; others suffer in silence; still others act out in compulsive or damaging behaviors. Anxiety, depression, and hostility may also be expressed in real or imagined physical symptoms. A study of rural women in El Salvador, for example, identified a remarkable range of psychophysiological "folk" symptoms that compare with standard definitions of anxiety states and depressive neurosis (Harrison, 1983). Many of these symptoms were attributed to stresses relating to reproductive patterns, child survival, and contraceptive use, over and above those engendered by other factors such as poverty and fears of the supernatural.

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[12] See, for example, Rainwater's (1960) study of lower-class couples in the United States.

[13] A major exception to the charge of neglect is represented by Bogue's (1983) review article. See also Schearer (1983).

[14] See the discussion of risks of pregnancy prevention in Winikoff and Sullivan (1987).

In analyzing the psychosocial consequences to women of contraceptive use and controlled fertility, it is helpful to identify the ways in which such behaviors affect women's perceptions of their performance of seven social roles: conjugal, occupational, maternal, domestic, kin, community, and individual (Oppong and Abu, 1987). Reproductive behaviors that intensify rather than reduce role strain or role conflict, particularly among those roles that a woman defines as most salient to her security and survival, are likely to be the most stressful. Negative outcomes may be reduced or avoided by adaptive mechanisms such as social support networks, economic resources, information, and structural changes that alleviate role strain or conflict and the resulting psychosocial stress.

Each of the four main elements in this model of reproductive behavior and psychosocial stress differs significantly among women according to numerous individual, group, and environmental characteristics: the nature of reproductive events and conditions (the potential "stressors" of primary interest in this model); the content and configurations of social roles; the nature and availability of intervening adaptive mechanisms; and the frequency, nature, and intensity of symptoms of psychosocial stress. Each element is also highly relevant to population and health policy in ways that cannot be pursued here. The four elements and their interconnections are intertwined in personal life histories and are deeply imbedded in social structures and ideologies of kinship, class, and caste. Further research on this topic requires not only culturally sensitive measures of psychosocial stress but also an analysis that is sensitive to the content in which sexual and reproductive behavior acquires meaning.

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Appendix
Background Papers

Reproductive Behavior and Child Survival Among Nineteenth-Century Mormons
Lee L. Bean, Geraldine P. Mineau, and Douglas L. Anderton
Psychosocial Consequences to Women of Contraceptive Use and Controlled Fertility
Ruth Dixon-Mueller
Family Planning Services Based on Reproductive Risk: A New Strategy of the Mexican Social Security Institute
James N. Gribble and Aurora Rabago
Mechanisms for the Association of Maternal Age, Parity, and Birth Spacing with Infant Health
John G. Haaga
Health Effects of Contraception
Nancy C. Lee, Herbert B. Peterson, and Susan Y. Chu
Maternal Mortality in the European Past
Katherine A. Lynch
On the Relationship Between Contraceptive Use and Access to Health Care in Developing Countries
Joseph E. Potter
Abortion and Contraception
Susan M. Rogers
Birth Interval and Birth Order Distributions over Thailand's Fertility Transition
Kua Wongboonsin and John Knodel
The Relationship Between Maternal Mortality and Fertility
Susan Zimicki